Principles and Practice of BURN CARE

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Principles and Practice of Burn Care

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To
My Parents who Made me
What I am Today

To
My Husband whose Constant Moral Support Gave me
The Base on Which I Worked

To
My Children, Rohan and Arushi whose Patience and Love Helped me
to Continue

To
My Father in Law who Inspired me
To Write this Book and his Constant Encouragement Helped me
Finish the Book
To
My Mother in Law whose Support Enabled me to Undertake this Venture
and last but not the least
To
My Sister for her Love and Encouragement

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Foreword

It gives me a great pleasure to write a foreword for this book entitled *Principles and Practice of Burn Care*, which has been ably compiled and edited by Dr Sujata Sarabahi, Dr VK Tiwari, Dr Arun Goel and Dr LC Gupta.

Sufferings of the World Wars have given some precious dividends of today. Efforts and contributions of Sir Harrold Gillies in various fields of Plastic Surgery including his invaluable contributions in burns outshine till this day. The unfortunate victims of burns are crippled, deformed, handicapped and disfigured and socially unacceptable. These poor patients are shoved into dark and smelly corners of the surgical wards of hospitals and left to die or scar in their own serum and sepsis. Being treated at general hospitals without skin grafting, most of them land up with horrible deformities and scars.

The magnitude of problem in a developing country like ours is a colossal challenge. We still do not have a central burn registry, which means that the magnitude of the problem is yet not fully understood, appreciated and defined. National Academy of Burns—India (NABI) conducted a survey over several years and found that about 7 million (70 lakhs) people get burnt annually, 7 lakhs need hospital admission, 2.5 lakhs get crippled with deformities and 1.4 lakhs die annually. The facilities for treatment are from meager to non-existent in some parts of the country. The progress has been no happy fluke, but a personal response to the challenge by a very few in the country. The research of some workers on the physiology and pathology of this trauma has given us valuable new insights; others have had the vision to see how new understandings could be translated into useful techniques, while yet others have responded to the clinical challenge by commitment of their lives to the delivery of burn care and to burn prevention.

Historically speaking, the first dedicated burn unit was started in India under my guidance in the Department of Burns, Plastic and Maxillofacial surgery at Safdarjung Hospital, New Delhi in 1963 and this department went on to become the largest burn care unit in Asia. This unit has been an example of commitment and dedication to the cause of optimum burn care in the country. Over the years, the details of treatment have been changing and facilities improved and advanced with progress in the world by various subsequent heads of the department. It is a matter of great pride that this book also comes from the same department where the story started in a barrack of a hospital designed for wartime casualties. The journey for this department has been long and arduous and I am happy to say that it has enjoyed the status of excellence all this while.

The editors are to be congratulated on recording in this volume entitled *Principles and Practice of Burn Care*, the views of several authors covering the entire country. This book has been divided into 7 sections, depending on the various situations in managing burns and has contributions from eminent specialists involved in burn care and research. Each section has chapters relevant to clinical management of the burn patient. The book is comprehensive and yet makes for easy reading. I am sure the students and doctors will find here the important practical information they need to give safe and thorough care to their burn patients.

I also hope that this book will be used in other developing countries especially by those who work in peripheral district hospitals and private medical practitioners with limited means. The basic essential to start a unit, is to have a committed staff who are ready to be spent for the sake of those in their care. The book will provide them with knowledge they need.

I wish this book a great success.

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Preface

Burns is the most serious injury ever inflicted on mankind. This appears to be the price of civilization which man is paying today. India is amongst the few countries of the world where the incidence of burns is showing a rising trend even though it is on the decline in the Western world. Management of burns is difficult and time consuming. Apart from being a costly treatment, it is also a heart-breaking experience for any specialist treating a burn victim and rehabilitating him back to the society is equally difficult.

Over the centuries, we have discovered how to use new technological innovations and scientific advances to the greater advantage of victims of burns, fire, bomb blast and other terrorist crimes. However, in view of the continuously changing circumstances, it becomes imperative for burn surgeons, physicians and other para-medicals to constantly learn to change and accept changes and keep themselves updated at all the times with the new skills and techniques in the management of burn patients. We cannot afford to remain silent, inactive or resilient even in the treatment of minor accidental burn cases which otherwise receive scant attention by the hospital staff possibly because they are not dangerous to life or due to lack of interest on their part.

In the 21st century we are learning to evaluate new products and technologies more realistically. In short we are re-discovering the wisdom of our ancestors and learning to value the efforts made by others so far in modifying and improving our methods and skills in burn care. Regular studies and continuous research in various fields have proven to be the right investment in improving every aspect of human life—so why not for burn injury?

Though a burn specialist treats burn patients, but the modern treatment of major burns depends upon multidisciplinary team approach which includes anesthetist, medical specialist, pulmonologist, nutritionist, physiotherapist and occupational therapist. Each of them helps in providing a holistic approach towards treating and rehabilitating a burn patient.

This book brings together the knowledge and clinical experience of many of the specialists from all over the country, who have made outstanding contributions in the field of burn care. An attempt has been made to deliver comprehensive information about the treatment of a burn victim in all its aspects from the experiences and expertise of these surgeons and physicians serving in the battlefield of burns, i.e The Burn Units; thus gaining and giving opportunities to learn from the best. This book emphasizes not only on the treatment and rehabilitation of burns but also the pathophysiology of burns and its prevention. It was not an easy task and required constant follow up to get others associated in producing this book.

I am thankful to all the contributors who are experts in their own fields for sharing their knowledge and experiences which have been reflected in their contributions, which I am sure will be beneficial to all the postgraduate and practicing burn surgeons who will be reading this book.

I have personally inserted photographs of many interesting cases in various chapters in addition to the photographs provided by the contributors in order to make the topic more interesting and easy to comprehend.

A book of this magnitude cannot be brought out single handedly. Dr Arun Goel, Dr VK Tiwari and Dr LC Gupta have been associated in editing this book and their help and support cannot be acknowledged in words. Without them, it would not have been possible to bring out this book which is complete in every sense, whether it is in the text content, pictorial depiction or practical case management protocol.

I am grateful to Dr SP Bajaj, who has been my guide and a father figure in burn management in India, for his valuable suggestions and encouragement at every step.

I am also thankful to my residents specially Dr Jeethendran Iyer for providing help in procuring the case photographs and Dr Geetika Khanna for providing the histological slides.

I must especially thank my publisher M/s Jaypee Brothers Medical Publishers (P) Ltd. especially Shri Jitendar P Vij (Chairman and Managing Director), Mr Tarun Duneja (Director, Publishing) for having recognized the merits of the presented material and to have come forward enthusiastically for publishing the book.

Lastly, I am thankful to my family for their moral support in helping me complete this book.

JAI HIND and Good Luck to my readers whoever and wherever they are.

Sarabahi

Sujata Sarabahi

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1 Section

General Considerations

Chapter

1

History, Epidemiology and Prevention of Burns

Ravi Chittoria

Burn Injuries represent one of the most important public health problems faced by both developing as well as industrialized nations today. Burn Injuries may be intentional or nonintentional but intent is sometimes difficult to determine. Burns or thermal injuries occur when hot liquids (scalds), hot solids (contact burns) or flames (flame burns) destroy some or all of the different layers of cells which form the human skin and even deeper tissues. For traditional reasons, skin injuries due to ultraviolet radiations or radioactivity, electricity and chemicals, as well as respiratory insults resulting from smoke inhalation, are considered as fire/burn injuries. Burns represent an extremely stressful experience for both the burn victims as well as their families. An extensive burn profoundly affects the patient's physique, psyche, financial situation and family. Patients suffering from extensive burn injuries frequently die while others suffer from painful and prolonged physical recovery. In different communities and countries, the etiological factors responsible for burn injuries vary considerably, hence a careful analysis of the epidemiological factors in different places is needed before the planning and implementation of a sound prevention program. This chapter highlights the history, epidemiology and prevention strategies for burns in both the developed and developing countries especially pertaining to Indian Scenario.[1,2]

HISTORY OF BURNS

"Those who fail to read history are destined to suffer the repetition of its mistakes".

"You have to learn what others have done because you won't live long enough to make all the mistakes yourself".

INTRODUCTION

Burn trauma is as old as the discovery of fire in the history of mankind. Medicine is built on the best of the past. Thus, a study of 'History of Burns' contributes to a review of accomplishments and errors, false theories and misinformation and mistaken interpretations. The history of burns, especially in India, is very fascinating. It teaches us where we started from, where we stand today, in what direction we are marching and guides us for the future.[1,2]

HISTORY OF BURNS IN THE ANCIENT AGES

World History

The philosopher Santayana said; "Those who cannot remember the past are condemned to repeat it". In an address to the Royal College of Surgeons, Churchill remarked; "The longer you look back, the further you can look forward". In considering the history of the treatment of burn injuries, one might quote Adam MacDougall, who in 1819 stated that "It would equally exceed the bounds of convenience and utility to particularize all the remedies that have been recommended in the treatment of accidents of this kind". Hippocrates (430 BC) used swine's semen, resin, bitumen and Oak bark solutions in the treatment of burns. Chinese (600-500 BC) used extracts of tea leaves. Smith papyrus (1500 BC Egyptians) used gum and goat's milk mixed with mother's milk and strips soaked in oil. Celsius (ancient Rome) advocated honey and bran. Glen (ancient Rome) described vinegar or wine in the treatment of burns[1-3] (Table 1.1).

Table 1.1: Burn treatment history

Neanderthal man	*Extracts of plants*
Smith papyrus (1500 BC, Egyptians)	Gum and goat's milk mixed with mother's milk
Chinese (600-500 BC)	Extracts of tea leaves
Hippocrates (430 BC)	Swine's semen, resin and bitumen Oak bark solutions
Celsius (ancient Rome)	Honey and bran
Rhases (9th century)	Cold water
Pare (1517-1596)	Excision and ointments
David Cleghorn (1792)	Vinegar and chalk poultice
Edward Kentish (1797)	Pressure dressings
Syme (1827)	Wool dressings
Lisfranc (1835)	Calcium chloride dressings
Passavant (1858)	Saline baths
Joseph Lister (1875)	Boric acid and Carbolic acid
Tomasalis (1897)	Salt water injections
Wallace (1949)	Exposure treatment
Keswani MH (1984, India)	Boiled potato peel (BPP) bandage

Indian History

Fire injuries in India have been common right from the ancient times. Suicide by fire out of insult was one of the common reasons. In Shiva Purana, it is mentioned that Lord Shiva's consort Sati had jumped into the sacred fire after being insulted by her father Daksha. Another reason for suicide by fire was 'Sati Pratha' a common tradition of committing suicide by widows. It is well known that Plastic Surgery originated in India in the Vedic Period about 3500 BC. Among the many distinguished names in Hindu medicine, that of Sushruta, the 'Father of Ancient Plastic Surgery' stands out in prominence (Fig. 1.1). Sushruta was the son of the sage Vishwamitra, contemporary of Rama of Ramayana. He compiled the surgical knowledge of his time in his classic 'Sushruta Samhita'. It is believed that this classic was compiled between 800 BC and 400 AD. Sushruta was the first to classify burns into four degrees: Singeing, Blister, Superficial and Deep Burns.[1-3]

Fig. 1.1: Sushruta at work

HISTORY OF BURNS IN THE MIDDLE AGES

World History

The British at the Battle of Crecy in 1346 were the first to use gunpowder and this development gave rise to many medical problems including those associated with the treatment of burns. The stimulus of the war experience initiated new and experimental types of treatment. In 1596, Clowes wrote a treatise on gun powder burns. He did not differentiate the depth of burns but rather described multiple types of treatment on different part of the body. He suggested oily dressings containing many drugs, did not open the blisters and was an advocate of bleeding. Ambroise Pare (1517-1596) described about excision and ointments. Fabricus Hildanus (1610), who wrote De Combustionibus, was the first to classify burns into three categories and also showed pictorially the early successful surgical release of hand contractures. Pare clearly described the differences between second and third degree burns that frequently developed contractures. Richard Wiseman in 1676 wrote several "chirurgical treatise" and discussed splinting to avoid contractures. He advised refrigerants or calefactive medicaments. Kentish (1797) described pressure dressings as a relief for pain and in his essay on burns promised to "rescue the healing art from empiricism and to reduce it to established laws". David Cleghorn (1792) used vinegar and chalk poultice for burns treatment. The use of ice and ice water for analgesia and the prevention of edema was explained by H Earle (1799) in his essay "the means of lessening the effects of fire on the human body". Syme (1827) used wool dressings. Lisfranc (1835) described calcium chloride dressings. In 1823 the Edinburgh Medical Journal published two papers on postmortem finding in two burn deaths. One patient had a gastric ulcer and another gastric congestion. Multiple lectures at this time were describing perforation of the stomach following burn deaths and there was increased interest in studying both the pathology and treatment of thermal injuries. Cotton dressing over burns was first discussed in a Glasgow Medical Journal in 1928. Readers were told how to apply dressing and were cautioned against changing them more than once daily. Dupuytren (1832), the brilliant French surgeon, made multiple contributions to burn research, including the documentation of the degree of injury to depth and the description of the phases of the post-burns course—irritation, inflammation, suppuration, and exhaustion. Sir George Ballingall in 1833 gave one of the best description of death from burn sepsis; "sinking in a hectic state, exhausted by a profuse discharge of matter from an extensive separating surface". In a land mark decision in 1848, the managers of the Royal Infirmary in Edinburgh designated one building for all burn cases and this became known as the burns hospital. Passavant (1858) gave concept of saline baths. In 1875 Joseph Lister recommended boric acid and carbolic acid for burn wounds in order to kill bacteria. In 1881 Tappeiner of Munich studied autopsies of burn deaths and recognized the concentration of blood, increased hemoglobin concentration and decreased blood and water volume. The appreciation by Tappeiner of burn pathology was a significant event. Thomasalis (1897) described use of salt water injection.[1-3]

Indian History

The history of burns in India in medieval period is that of decline in India and lull in European countries. The natural feeling of fear for operations grew and love for medicines increased. The governments prohibited and neglected the practice of surgery. The famous Buddhist scripture Mahavaggaa Jatak enjoined strict prohibition of surgery and extended the terror of hell both for the surgeon and the patient. 'Manusmriti' prescribed special rituals by way of purifications of surgeons, who were looked down upon as unclean. Later during the Rajput, Muslim and Maratha periods, Ayurvedic (Hindu) and Unani (Arabic) systems of medicine were practiced by Vaids and Hakims respectively. The Vaids were purely physicians and the practice of surgery was restricted only to incision of abscess, dressing of wounds and treatment of bone and joint injuries by barbers and bone-setters. Not much information is available in literature related to burn injuries.[1-3]

HISTORY OF BURNS IN THE MODERN AGE

World History

In 1905, Sneve noted the importance of intravenous saline for resuscitation and wrote an article in the Journal of the American Medical Association advocating early skin grafting. The understanding of burn pathology took a great leap forward when Underhill (1930) studied a group of patients burned in a theater fire in 1921. He analyzed the content of blister fluid and determined that burn shock was due to fluid loss and not toxins (a popular theory of that time). The Coconut Grove Fire in Boston in 1942 resulted in extensive studies by Cope and Moore on the diagnosis and treatment of burn shock. Controversies over the best method to treat the burn wound persist to the present day. Dressings remained popular until Wallace in 1949 advocated exposure treatment for burns of the face, buttock and perineum. Formulas gradually evolved to calculate fluid losses. Evans (1952) used burn skin surface area and weight as the principal variable. The Brooke formula was a modification of the Evans formula and utilized salt, colloid and water. Moyer and Associates (1965) recommended only Ringer's lactate solution, yielding alert, oriented patients even when the body surface area burn was large. The pattern of triage of burn patients changed dramatically in the 1960's. The US Army Research Unit was initially guided by Pulaski and Artz, who stressed patient care as well as clinical and laboratory burn related research. Critically injured patients, civilian and military, were referred to the center and the concept of the multidisciplinary burn team evolved. In 1962 the Shriners fraternal organization in the United States began development of three burn centers affiliated with universities and dedicated specifically to the care of burned children. These centers served as role models that have completely altered burn care around the world. Their superb survival statistics made others realize that patients with large burns were not automatically doomed but had a chance of survival if cared for in a specialized center. Laboratory and Clinical Research in burn injury improved other areas of trauma care as well. It became obvious that the burn patient with multi-system involvement was the perfect trauma model. The universality of the burn problems provided a reason for better international cooperation and exchange of ideas. In 1960, the first international congress on research in burns was held in Washington, DC. At the second meeting in 1963, The International Society for Burn Injuries (ISBI) was founded and it has been conducting meetings every four years. The American Burn Association was founded in 1968. The society's membership was unique in that it consisted of all the members of the burn team, including non-physicians (Dieticians, Physical and Occupational therapists, Nurses and Psychologists).[3]

Indian History

Burns became alarmingly frequent in India in the modern age of industrialization. To pour kerosene over the clothes and then set them on fire is the most common form of committing suicide or murder. There are also innumerable instances of accidental burning caused by clothes catching fire on low stoves or open gas rings; the loose flowing end of 'saris' and 'dupatta' of Indian women increases this danger enormously. Open fires in winter are an added hazard. Acid burns of the face are frequently the result of blind jealousy. Suicidal burns amongst Hindu women are usually fatal because of its extent. Practice of tradition of 'Sati Pratha' declined in this period. Most of the burn cases in India are treated by General Surgeons and General Practitioners in small cities and rural areas because Plastic Surgeons are few and settled mostly in Metro cities. These General Surgeons and General Practitioners have negligible facilities and have no forum to exchange, enhance or update their knowledge. The initial local treatment on admission to hospitals varies according to the area involved in the burns—face, front of chest and abdomen are routinely treated by the exposure method. Burns of extremities and fingers alone are dressed. The majority of hospitals do not have the 'Stryker Frame' and hence only circumferential body burns are dressed. Resistant strains of organisms have increased in recent years due to excessive use of antibiotics. Surgeons are not in favor of keeping grafts exposed from the start. Skin grafts are dressed with pressure pads for a week and then kept exposed. To keep the donor area exposed was attempted by many but were not satisfied by this method. In fact, dressing of the donor area is retained for a fortnight during which the part heals. Severe cases of contractures of the flexor creases like the neck, axilla and elbow are released and skin grafted. Free grafts are preferred to tube pedicles as the relief is quick as well as complete.[4]

In India many Plastic Surgeons have contributed in the development of Burns Unit and management in modern era. Dr Noshir H Antia, the 3rd generation trained Plastic Surgeon under Sir Harold Gillies was the first surgeon to start a unit at Grant medical College and associated JJ Hospital, Mumbai in 1959. In 1964, Tata Trusts provided a substantial grant to carry out two projects–one on Leprosy and other on Burns. In 1963, Safdarjung Hospital, Delhi started a department of Burns, Plastic and maxillofacial surgery with Dr JL Gupta as its head. Professor Antia felt that they should have a forum and thus, a Burn Association of India (BAI) was formed at JJ Group of hospitals, Bombay in 1972. Dr MH Keswani was the Secretary. Later under leadership of Dr Keswani the Burns Association of India (BAI) flourished with contributions like Boiled Potato Peel (BPP) dressings, prevention campaigns by way of radio and TV talks, small documentary ads, 'School Education Program' in burns etc. "Pour water on Burns" has received worldwide popularity. 'AGNIVARTA' is a quarterly publication of 'Burns Association of India' devoted to the problems concerning Burns. Burn injury is a disease of the poor at least in our country. Dr Keswani gave various modifications to reduce the cost of burns treatment like common home made curd (called yogurt in the US) as a de-sloughing agent, Butter Milk Diet (BMD) as cheap high calorie high protein diet, X-Plasty of Vartak as a simple operation of release of contracture, Vartak's wheel as mesh graft dermatome, cellophane dressing for donor area, simple operation cum bath trolley, etc. Dr Keswani has to his credit the setting up of the first children's burn unit in India and India's first skin bank at Bai Jerbai Wadia Hospital in Parel, Mumbai in 1975 and 1978 respectively.[5-7]

In 1990, Prof J L Gupta convened a meeting at Pragati Maidan, New Delhi. A very active and detailed discussions and consultations were held amongst Prof JL Gupta, Prof Mathangi Ramakrishnan, Prof J K Sinha, Prof S P Bajaj, Prof S K Bhatnagar, Prof R K Keswani, Prof Siti Roy Choudhary, Prof PK Bilwani, Prof Madhuri Gore, Prof D L N Prasad, Prof Swarna Arora and Prof Anil Chadha and many others. This historic and significant meeting led to the birth of National Academy of Burns - India (NABI). NABI mainly aims at preparing and making available a suitable appropriate and latest information relevant to all those concerned with burn care. Recognizing the services of Prof J L Gupta in the field of burns in the country, he was unanimously named as the Patron of NABI. Prof S N Sharma was the choice for the founder Presidentship. Dr S P Bajaj became the founder Secretary. The academy brought out an annual journal named 'Indian Journal of Burns' with Dr Rajeev B Ahuja and Dr Arun Goel as editor and assistant editor respectively. They worked for five years (1993-1997) before passing on the task to others. The first volume was brought out in 1993 and the journal is being regularly published. While NABI is an association of burn care professionals (doctors, nurses, therapists etc.), BAI even included persons like businessmen, old recovered burn victims, etc. who were ready to serve the cause of burns in their own way.

Dr JL Gupta gave guidelines for planning a Burns Unit.[8] Subsequently many Burn Units have been developed successfully in various Institutions and Medical Colleges like Burn Unit in the Department of Plastic Surgery at Kasturba Medical College, Manipal, Karnataka, IPGME&R, Kolkata etc. Of all the existing Burns Units in India, special mention is made of Burns Unit of Safdarjung Hospital, New Delhi which is the largest burn unit in the country and which has been modernized and equipped with all modern Burn care infrastructure facilities. 'Early Tangential Excision and skin grafting', 'Rescuer Burns', 'Policy of antibiotics in Burns' etc. has been popularized extensively by the Burns Unit of Manipal, Karnataka.[9,10]

In India, Burn injuries due to fireworks on the occasion of 'Diwali' are very common. Safety tips related to Fireworks have been explained by Anjali Burma.[11] The Government health system is not able to keep pace with population growth in recent years. Although major burn accidents are decreasing in relation to the population growth, the actual number of burn cases is on the increase. Gradually more and more teaching hospitals started separate Burn wards. Possibly about 60% of them are managed by the General Surgery services and the rest by a Burn and Plastic Surgeon. The requirement for more burn beds encouraged many non-government groups into establishing burn management centers. Many of these centers have well equipped wards, trained support staff and enthusiastic Burn Specialists. These specialists have been instrumental in maintaining the prevention and rehabilitation programs concerning burns.[9,10]

RECENT ADVANCES

What have been the outstanding features of burn injuries in the last twenty five years? It is the better understanding of burn pathophysiology that is the most remarkable feature of last quarter of a century. The second important feature is the realization that the care of Burns is a team effort, the leader of the team being the one, who can lead the team most inspiringly, a person with vision, with dedication and with tenacity. It would not be fair to single out any one individual for all the advances. Half a century ago, the commonest cause of death following a burn greater than 20% was surgical shock, at least in India. With a better understanding of post burn surgical shock it is possible to treat this rationally and efficiently. The mortality from this cause has reduced considerably. Generalized infection of the tissues (septicemia) is the single most common cause of death following burns, at present. Better drugs and their wide-spread availability have helped us to control infection to some extent. However, while these drugs are useful, they have many undesirable side effects, some of them lethal. Moreover, the cost of these drugs is prohibitive. They have encouraged resistant strains to develop and they have put the clock back in many instances. Post burn sequelae need correction. These have been satisfactorily corrected by reconstructive surgeons and in many instances by interested general surgeons. Early post burn rehabilitation has been encouraged, but much progress needs to be made in this direction in this country. When the hands are burned, it is a severe blow to the person's economic viability. Better methods of rehabilitation need to be developed. Unfortunately over the last 25 years, not much progress has been made in this area. It costs approximately US $1,000 per patient per day to provide satisfactory care in the Western world. This is clearly not possible in India and in many of the developing countries. Prevention of burns is the only logical solution. This is not easy and is time consuming. Easy or not, we have no option(s). We must prevent burns. Fortunately the Burns Association of India, which was formed in 1972, has been doing precisely that. Burns Prevention has to be propagated zealously by everyone who is able to do so, especially Doctors, Nurses, Paramedical workers, Social workers, teachers, surviving burns patients, and the media — in fact it should be a national responsibility. India is a country with tremendous problems-those of population explosion, education, malnutrition, unemployment and many more. Our hopes lie in a unified approach to work together and solve some if not all of these problems.[12]

EPIDEMIOLOGY OF BURNS

"I keep six honest serving men; they taught me all I know. Their names are 'WHAT', 'WHY', 'WHEN', 'HOW', 'WHERE', and 'WHO'."

INTRODUCTION

Epidemiology has been defined by John M. Last in 1988 as: "The study of the distribution and determinants of health-related states or events in specified populations and the application of this study to the control of health problems". Epidemiology began with Adam and Eve, both trying to investigate the qualities of the "forbidden fruit". Epidemiology is derived from the word epidemic (epi = among; demos = people; logos = study), which is a very old word dating back to the third Century BC. Burn injuries and their related morbidity, disability and mortality represent a public health problem of increasing importance in developing countries. Developing countries have a high incidence of burn injuries, creating a formidable public health problem. Epidemiological studies of burn injuries are a prerequisite for effective burn prevention programs because each population seems to have its own epidemiological characteristics and knowledge of the epidemiology of burns is needed to select target groups for preventive actions.[13]

BURN REGISTRY

The American Burn Association (ABA) commissioned Jeffrey T Suffle and Byron of the University of Utah Health Center, Salt Lake City, Utah, USA, to develop a computer program to assist burn units to collect data useful for patient care and prevention. The ABA graciously permitted the International Society for Burn Injuries (ISBI) to distribute a version of this program, adapted for use internationally, to ISBI members free of charge.[14]

USES OF BURN DATA

Burn data are useful to:

1. Define the burn problem.
2. Determine the most common ways that people are burned.
3. Evaluate the effectiveness of medical care for burn patients.
4. Identify which burn hazards should be targeted by burn prevention programs.
5. Examine the economic impact of burn injuries and burn treatment.[14]

SOURCES OF BURN DATA

The sources of burn data vary, depending upon the severity of the burn injury:[14]

i. *For burn deaths:* Collect information from hospitals and local, regional or national vital statistics registries. Many people die immediately of fire and burn injuries and do not receive medical care.
ii. *For serious burns:* Collect information from hospitals; decide whether to collect data only on burn patients admitted to the hospital, or on all patients seen at the hospital. If there are several hospitals treating burn patients from the defined population, collect data from all these hospitals. Referral patterns may distort the true profile of burns in that population.
iii. *For less severe burns:* Collect information from health clinics, doctors' offices, visiting nurses or any other medical treatment service. A good sampling plan will make this task easier.
iv. *For burns of all levels of severity occurring in a community:* Collect information through a community survey, asking residents in face-to-face or telephonic interviews to recall circumstances of burn injuries suffered by family members. Questions about burn injuries could be added to a larger multipurpose community survey.
v. *For burns associated with building fires:* Contact the local fire service for information about fires in which people were killed or injured. Remember, this is not a good source for clothing ignition burns, scalds, contact, electrical or chemical burns.

STANDARD CODING OF BURN INJURY DATA

Most countries use the World Health Organization's International Classification of Disease (ICD) Codes for coding data on hospital discharge records, although there are other coding schemes. For example, the Nordic countries use a coding scheme called NOMESCO. The ICD system contains two sets of codes for burn data. The first set classifies what type of fire or burn injury was sustained:[14]

Code	*Type of Burn*
940-949	Burn injuries
986-987	Smoke inhalations
692.71	Sunburn
910-919	Superficial injuries (friction burns are among these codes)

The second set of codes is the ICD E-codes (External Cause of Injury), which classify the causes of burn. These are four-digit codes, with the fourth digit often capturing important burn information:[14]

Code	*Cause of Burn*
E890-899	Burns caused by fire and flames
E923	Accident caused by explosive material (Includes fireworks and explosive gases; not all injuries coded here are burns)

E924	Accident caused by hot substance or object, caustic material and steam
E925	Accident caused by electric current (burns or electrocution)
E926	Exposure to radiation
E958.1, .2	Suicide (by burns or scalds)
E965-968	Homicide and assault (965.6, 967, 968.0, 968.3 related to burns)
E988.1, .2	Undetermined if accidentally or purposely inflicted
E990, 996	War

NATURE OF DATA NEEDED TO STUDY BURN EPIDEMIOLOGY[14]

How Big is our Burn Problem?

Determine the number of people who have been burned. Do not ignore burn deaths, especially those who die before coming to the hospital. Counts of non-fatal burns are made easier in areas where computerized hospital discharge records are coded using the International Classification of Disease Codes or another standard coding scheme. Count the number of patients with a burn injury diagnosis, coded using 'type of burn' codes.

How are our People Getting Burned?

Determine the number of people burned by specific causes of burns, such as clothing ignition or scalds. Collect information which will permit patients to be classified using 'cause of burn' codes. Accurate, useful coding depends upon accurate, useful information in the medical record. This includes details about the circumstances of the burning, ignition sources and other product involvement, where the event happened and the activities of the people involved and demographic information about the patient. This often requires an interview with the patient, a family member or someone present at the burn episode. Make sure that this information is written in the medical record, coded and, whenever possible, entered into computerized hospital discharge records.

How can we Prevent Burns?

Analyze data describing the common causes of burn injuries in the population to identify high risk groups such as young children and high-risk activities, such as meal preparation.

How can we Evaluate the Success of our Prevention Programs?

The precise nature of the data required depends upon the prevention program and the research design of the evaluation.

Information about Larger Populations

It is important to know certain characteristics of the defined population, whether this be a village, a region or a country. Important characteristics are: the number of people in the population and the age distribution of these people. If your country collects and publishes census data, these provide an excellent source of information to use to describe risk factors for burns in your population.

Burn Rates

Burn epidemiology uses rates of burn injury or deaths. A rate is calculated by dividing the number of burn injuries sustained during a specified time period by the number of people in a population during that same period. It is expressed, for example, as 'burns per 100,000 populations per year'. One must use burn rates to compare the burn problem in communities or countries with different sized populations. A densely populated community may have many more burns but a relatively lower burn rate than a sparsely populated region. A lower burn rate signifies a lesser burn problem. It is important to know the age distribution of the defined population because burn risk is age-related. A population with a lower median age will have higher rates even if its adults have an equal or slightly lower risk than a population with a higher median age.

Usefulness of Burn Data from a Single Hospital

If only one hospital treats patients with serious burns in a defined population and if one is interested only in serious burns, the vital statistics registry and this hospital are the two appropriate data sources for fatal and serious burn injuries, respectively, for that population. If the hospital is one amongst many serving a single population, one cannot draw any conclusions about the population's burn problem by studying only one hospital. However, even without collaboration with other local hospitals, it can be valuable to analyze burn data from a single hospital. Individual hospitals can provide useful information about a specific circumstance which causes burns and can provide 'cases' for a 'case-control' study. One cannot, however, draw conclusions about the population's burn problem.

EPIDEMIOLOGY OF BURNS IN INDIA

National Burns Registry

One of the major activities of The National Academy of Burns-India (NABI) is to maintain a National Burns Registry. Various data is collected from centers engaged in management of Burns. This is, however, a voluntary effort and has so far not reached to all places. In a few years with continued efforts and support from all areas, National Burns Registry of NABI will be able to truly reflect the scene of burns problem in India.[15]

Extent of Problem (Mortality and Morbidity)

Over 100,000 people are affected by burns in India annually and 20,000 of them die according to rough estimates.[16] In 1991, the reported deaths were 22,306. A survey of almost 3500 hospital admission over the past few years supplemented by intermittent published reports over the past 30 years indicated a mortality rate of between 25% and 49% for adults and between 6% and 20% for children (0-10 yrs old).[17] Ten percent of all accidental deaths and seven percent of all suicides in India are caused by fire. Although incidence of burn trauma is on the decline in the developed nations, it is still rising in India and is second largest killer after road traffic accidents. Thousands of victims of burns are being mutilated, disfigured and handicapped every year.[18] The cost of burns is both visible and invisible. In India, the cost of burns ranges from $25 to $300 a day. To this may be added the loss of wages of the patient and of those who have to be away from work to care for him or her during hospitalization. The other visible cost is in the scarring and deformity, of loss of hair and body- pigment and alterations in skin texture. There is also a loss of physical skill which leads to demotion at the place of work and makes one less fit or unfit in domestic life. The invisible cost is in the pain the patient undergoes, both physical and emotional, the emotional trauma of itching, of scarring and intolerance to heat. Neither the trauma of patient's psyche nor the imperceptible alterations of the patient's personality can be measured. And then, there is loss of time—of time lost from studies, of vanished opportunities in social life, like marriage or career promotion. And finally, one has to remember the cost to the patient's family–to the husband who loses a beloved wife, to the child who becomes motherless, to a young wife who becomes widowed and to the aged parents who loose their support. In India, society is structured differently and the cost borne by the patient's family is truly immense and immeasurable.[19]

Age and Sex Distribution

The age distribution revealed by various studies in India shows that more than half the cases are aged between 21-40 years. In children, the lack of coordination and unawareness of dangerous substances play an important role in the occurrence of burns. High incidence among young adults may be explained by the fact that they are generally active and exposed to hazardous situations both at home and at work.[20-22] Male predominance in the younger age group (5-10 years) might be explained by the inquisitive and exploring nature of boys of this age. As regards sex distribution, the female preponderance in the age group 20-40 years concurs with reports from other developing countries and might be explained by the involvement of females in domestic activities and dowry deaths too. However, figures from industrialized countries are clearly lower than those reported from developing countries. This may be due to the relatively lower percentage of occupational and recreational burns or due to better home safety with safer cooking and heating devices in industrialized countries.[23-25]

Sociocultural Factors

Sociocultural factors are among the major causes of different age and sex predisposition of developing countries like India compared to other developed nations. The majority of the patients belong to low socioeconomic status (69.6%), while 21.8% are from middle and only 8.6% are of high socioeconomic status. 42.6% belong to an urban area while the rest are from rural areas. Arrival time to hospital after the incidence is variable, only few patients (9.7%) arrive within six hours of the incidence. The majority (61.8%) reached the hospital between 6 and 24 hours; those reaching the hospital after 24 hours contribute about 28.5%.[26-28]

Etiology

An extensive study was done by the department of Burns, Plastic and Maxillofacial Surgery, Safdarjang Hospital, New Delhi.[29] Analysis of the causes of 9927 burn cases is given in Table 1.2.

Table 1.2: Showing incidence of different types of burns

Causes of Burns	%
Dry Burns	51.64
Scalds	33.35
Electric Burns	3.59
Chemical Burns	2.97
Petrol Burns	2.36
Coal Tar Burns	0.79
Industrial Burns	0.75
Miscellaneous	4.55

It is seen that the vast majority in these are dry burns, by direct contact with either flash or flame. The second most common are scalds. This is because of conditions which are peculiar to the villages in our country. Most of the cooking is done at the floor level with open fire. Stoves, coal and wood are commonly used. Often large families live and cook in the same room. If there is a kitchen, it is small, crowded and ill-designed. The storage shelves lie above the chulla/angithi (a kind of open oven used in India) or stove, so that while getting up to pick up an article from the shelf, any loose garment can easily catch fire. The lamps used for illumination are made of an ordinary glass bottle or tin can, with cotton wick and kerosene oil. They are either kept on the floor or on any shelf. They can be easily upset and result in an oil fire. The majority of the population lives in villages in thatched houses which easily catch fire. Accidents at home form the major bulk and the common causes are:

1. Loose clothing like 'sari' and 'dupatta', mostly made of synthetic, inflammable material.
2. Spilling and spraying of kerosene oil from pressure stove is more common than actual bursting of it. The jet of oil under pressure catches fire immediately.
3. Hot food stuff and liquids carry a constant danger to children and infants, particularly in over crowded rooms. They often fall in the steaming food, cooking utensils and hot water buckets and tubs.
4. It is common to heat water in a tin with its lid in position. As the lid is handled, the steam under pressure inflicts severe scalds of face, neck and upper chest wall.

After flame and scalds, electrical burn is the third most common cause of burns in India. The electric appliances used are substandard and are used without any regard to safety measures. Electrical burns while flying kite is very common in India especially in young children.

Circumstances of the Injury

There has been a change in the pattern of burn injuries as far as circumstances of the burn injury are concerned. Recent study shows that majority of burn injuries, i.e. more than 85% occurs in homes. Kitchen-related activities are most often responsible for burns in domestic setup. Flames represent the most common agent of burn injuries (75.1%). Among the flame burns, kerosene pressure stove is the most common source. Chimney and Liquid petroleum gas (LPG) burns rank the second most common source of flame injuries. Most of the burn injuries are accidental (67.7%), followed by homicidal (13.4%) and suicidal (18.9 %).[30]

Epidemiology of Pediatric Burns

Pediatric burns differ from adult burn injuries in many aspects and need separate consideration. Only few pediatric burn units exist in India. Most of the children with burns are treated in a common burns unit. Epidemiological studies on childhood burn injuries in India are very scant. Information on the exact magnitude of burn injuries has not been well documented. It is known, however, that burn hazards are present in the daily life of children. Household activities, including cooking are expected of girls early in their lives. According to one of the epidemiological study of pediatric burns, various risk factors attributed to burn injuries in children are low socioeconomic conditions, poor living conditions, illiteracy, overcrowding and floor level cooking, on either kerosene pressure stove or an open fire. These pressure stoves are sometimes of shoddy construction and commonly malfunction leading to explosive disintegration. The overcrowded conditions cause young children to play in the vicinity of the cooking area leading to scalds from spilled hot fluids or foods. Pre-existing diseases and malnutrition afflict a considerable number of patients, particularly children injured in slum areas, thereby ensuring that patients have a reduced ability to compensate for the physiological stress inherent in a severe burn. The ridiculous first aid measures like application of ink, cow dung, hen's blood, haldi (turmeric), honey and other home made remedies, surprisingly, are still frequently used. Delay in transportation and initiation of treatment due to lack of education, lack of transportation facilities and lack of economic support are still prevalent.[31] According to the recent study of 500 pediatric burns, majority (24%) of burns occur in children between one and two years age group.

Male to female sex ratio was 1.38:1. Burn injury occurred predominantly during winter. Most common type of burn was scalds which occurred mainly in domestic circumstances. Mortality rate was 10.4%. Mortality rate was high in patients having more than 40% TBSA involvement. Parental occupation, family size and the first aid did not affect the mortality rate.[32]

PREVENTION OF BURNS IN INDIA

The only thing more expensive than education is ignorance

Prevention is better than cure

INTRODUCTION

Burn injuries are universal and incidence is on the increase with the complexities of modern living and industrialization. As most of the burns occur either at home or at the place of work, it becomes important to know about Burn Prevention. In order to bring down the incidence of burn, education programs are oriented to bring the changes of culture pattern, change in the way of life, change of rules and regulations at work place, change in dangerous substandard gadgets and change of habits. Prevention does not mean only to reduce the incidence but also to reduce the depth and severity of the burn.[33]

RISK FACTORS

Before preventive measures are discussed it is important to know about the risk factors especially in Indian scenario. Various risk factors are as follows:[34]

At Home

In India most of the burn injuries occur at home, especially in the kitchen. Various risk factors are floor level cooking, substandard kerosene pressure stove, loosely worn garments (saris and dupatta), low level electric plug points, long over hanging flex of kettle, overhanging table cloth over which hot food or beverages are kept, carelessly kept match boxes within reach of children, substandard pressure cookers, etc.

At Work

Inadequate safety measures, overwork and fatigue are common risk factors at work place.

At Play

During Diwali, Indians play with fire crackers and celebrate the festival with lights. Inferior quality of crackers and carelessness during their bursting are important risk factors.

RESEARCH DESIGNS FOR BURN PREVENTION

Given below are the broad outlines of four research designs which may be helpful in planning and implementing burn prevention programs and their evaluation.[35,36]

Nonexperimental Descriptive Study

Purpose

To determine dimensions of a serious burn problem in a defined population.

Tasks

1. Define the population of interest, determine numbers and age distribution, if possible.
2. Decide on a time period. Include at least one year, since in most locations, burn injuries have seasonal variations.
3. Decide on the level of burn severity. Include all deaths and people hospitalized for a burn injury and may be those treated and released from hospital or clinic.
4. Identify all hospitals treating burn patients in the defined population. Request permission to review medical records or request hospital staff's help to provide data.
5. Use a standardized burn form to collect all available information. Record as much information as is available on the circumstances surrounding the burn episode. The ISBI Burn Registry computer software is a useful tool for collecting and recording burn data.
6. Prepare report to include rates:
   a. Death rates [(no. of deaths/no. in population) × 1000,000], expressed as deaths per million persons
   b. Injury rates [(no. of injuries/no. in population) × 1000,000], expressed as injuries per million persons.

   Calculate rates for the following categories:
      i. Age groups
      ii. Gender
      iii. Type of burn (flame, scald, other)
      iv. Severity (depth and extent of burn).
7. Identify the most common circumstances and/or products causing injury (e.g., pressure stoves, clothing, ignition).

8. Plan a prevention program to address these problems. A review of literature may reveal which strategies have been effective.

Preexperimental Design: One Group Pretest-Posttest Design

Purpose

To document change associated with a prevention program.

Cautions

There is no control for natural time trends, societal forces and factors other than the intervention which influence burn rates.

Tasks

1. Conduct the descriptive study to determine the burn problem in a defined population. Determine rates for those types of burn injuries and those age groups expected to be affected by the program.
2. Design or adapt a burn prevention program which addresses a significant burn problem.
3. Repeat the descriptive study and compare burn-type and age-specific rates before and after the intervention.

Note: If the prevention intervention has already been instituted, as it often is with safety standards or regulations, the descriptive studies of burns before and after implementation are retrospective and may be conducted simultaneously. Burns are relatively rare occurrence and are known to have yearly fluctuations. For this reason, it is well to collect data for several years before and after intervention.

Quasi-experimental Design: Non-equivalent Control Group Design

Purpose

To measure the effectiveness of a burn prevention program.

Tasks

1. Conduct the descriptive study to determine the burn problem in two communities. These communities should be comparable in size, age distribution and socioeconomic variables.
2. Select a burn prevention program, proven to be effective in other locations, which addresses a burn problem shared by both defined communities.
3. Randomly select one community as experimental and one as control. Conduct the prevention program in the experimental community.
4. Repeat the descriptive study in both populations and compare burn-type and age-specific rates. A successful program would be indicated by significantly lower burn rates in the experimental community with rates essentially unchanged in the control community.

Experimental Design: A Case-control Study

Purpose

To measure the effectiveness of a burn prevention program, for example, whether a new inexpensive kerosene stove, recently introduced into a community, has reduced the likelihood that someone will be burned.

Tasks

1. Identify 'cases', that is, patients treated for burn injuries caused by stoves.
2. Interview the patient or family member to determine the type of stove used by the patient. Determine fuel source, whether or not stove is the 'new design'.
3. Select appropriate 'control' for each case. This is the most difficult task of a case-control study, to find persons comparable to the case in all things except the type of stove used in the home. It is well to find multiple controls for each case, drawn from different categories. For instance, a hospital 'control' could be a person admitted to the hospital the same day for an illness rather than an injury, who is the same gender, same socioeconomic status and in the same age group as the burn patient. A neighborhood 'control' could be a person living on the same road, within a certain number of houses, who is the same gender, same socioeconomic status and in the same age group as the burn patient.
4. Interview each 'control' to determine the type of stove used. Determine fuel source, whether or not stove is the 'new design'.
5. Analyze data using simple 2 × 2 table, categorizing subjects along two axes: positive or negative for burn injuries associated with stoves and positive or negative for exposure to 'new stove'. Success would be achieved if those using the new stove were significantly less likely to be in the burn category.

EVIDENCE THAT BURN PREVENTION CAN SUCCEED

- Throughout history, whole cities have burned down, cities like Rome, London and Chicago, to name but a few. Today, such massive fires are rare; when they do occur, they are associated with war and natural disasters. There are many reasons why whole cities do not burn down: More fire-safe construction methods and materials, fire-safety-related building codes and standards, professional fire services and water delivery systems.
- Fewer children are being burned when their clothes catch fire. There are many reasons: sleepwear flammability standards, the introduction of new, less flammable fibers and fabrics and more close-fitting designs for girls' clothing. This pattern has not been seen in all countries, particularly not in those countries like India where girls and women commonly wear flowing dresses (saris) made of cotton and rayon.
- Fewer people in the USA are dying in residential fires, due in part to the widespread adoption of smoke detectors which warn residents of deadly smoke.
- Electrical burns of the mouth were all but eliminated in Denmark when a defective electrical cord for a popular vacuum cleaner was recalled and replaced.[37]

COMMON ELEMENT IN SUCCESSFUL BURN PREVENTION PROGRAMS

A consumer product or the environment is made safer for everyone by using prevention programs. Although it might seem easier to teach people to act safely in an unsafe environment, it does not usually work that way. Put your energy into making a safer environment. Burns can happen to anyone, but some groups are at greater risk: the very young or very old, the poor and people with disabilities or who are impaired by drugs or alcohol. These groups are the hardest to reach with educational messages or are less able to adopt safe practices, even if they hear the educational messages.[37]

BASIC PRINCIPLE UNDERLYING BURN PREVENTION STRATEGIES

Keep people separated from excessive heat. This may appear simplistic, but it is fundamental. A person cannot be burned unless a person and a heat source come into contact.[37]

- *Separate with a barrier:* For example, insulate the outer surfaces of stoves and heaters to prevent contact burns; install fences to keep trespassers away from electrical power stations; use sun block creams, umbrellas and roofs to protect people from sunburn.
- *Separate by location:* For example, keep children out of the kitchen or away from the cooking area.
- *Separate by time:* For example, permit trucks carrying volatile fuel to be driven through tunnels only during hours of low traffic volume.

SEQUENCE OF TASKS FOR BURN PREVENTION PROGRAMS

There are six major tasks within a burn prevention program:[37]

1. Document the problem.
2. Set goals.
3. Consider possible strategies.
4. Design the intervention.
5. Implement program.
6. Evaluate program.

Above tasks can be exemplified by study program conducted in India by Injury Prevention Workers to protect the people of India from burns caused by fireworks; a secondary success was increasing the use of cool water as first aid for burns.

Document the Problem

Determine the size and nature of the burn problem in your community. Make sure you get good information about what causes these burns and what products and circumstances are involved e.g., in India, researchers found out that the 'cone fountain' fireworks were involved in most of the injuries sustained during Diwali, the festival of lights.[38]

Set Goals

Decide what you want to accomplish in your prevention program. Have single and specific goals, not multiple and broad ones, e.g. in India, researchers decided to prevent injuries caused by 'cone fountain' fireworks.

Consider Possible Strategies

Use the 'public health model', following the example of public health specialists who consider three factors (Agent, Host and Environment) when trying to eradicate diseases. Energy (be it thermal, electrical, chemical or ultraviolet radiant energy) is the 'agent' for the burn injury. Host is person at risk. Environment includes both social and physical environment. Here is an example how planners in India have addressed host, agent and environmental factors when designing their programs:

Host (person at risk)	Teach proper use of fireworks
Agent (energy source)	Redesign 'cone fountain' fireworks or ban them
Environment (social and physical)	Restrict sale of these fireworks to professionals and/or restrict locations where they can be used

Design the Intervention

Decide how your goal will be accomplished. Be specific about what is to be done, who is to do it, in what time frame, with what resources, e.g. in India, researchers decided to mount a media campaign during Diwali, to teach people to handle 'cone fountain' fireworks safely and to pour water on burn.

Implement Program

Gather the necessary resources to implement your program and get to work. This means finding financial support if necessary and hiring people or finding volunteers to perform appointed tasks and to monitor progress. It means regular review of progress and brainstorming on how to correct elements of the program that are not working. It means hard work. In India, researchers got financial support and technical expertise from a local insurance company and advertising firm which made television spots about safe handling of 'cone fountain' fireworks and about cool water as first aid for burns. Television spots, print advertisements and brochures were prepared. The television ran the spots just before and during Diwali.

Evaluate Program

Evaluation is the key to progress. This is true of surgical procedures, infection control, skin replacements and prevention practices. It indicates what is effective and what is not, what should be duplicated, what should be redesigned and what should be dropped. Evaluation is complex, but easier when the goals of the program are very clear. The bottom line is fewer burn injuries. Evaluators try to determine if there are fewer burns after the program, linked to measurable changes in behaviors, products or the environment. In India, researchers found no significant difference in the number of people injured by 'cone fountain' fireworks during Diwali after the media program. However, they found significant increases in the number of patients who used cool water for first aid for burns. Thus, this program had mixed results.[38]

GENERAL GUIDELINES FOR POTENTIALLY EFFECTIVE BURN PREVENTION ACTIVITIES

Burn prevention efforts must be tailored to the characteristics of specific burn risks. It is not possible to prevent all burns simultaneously. Burn prevention specialists must be patient and systematic, addressing one type of burn at a time. The best strategy to prevent scald burns will not reduce the number of flame or chemical burns.

Kerosene Lamps and Stoves

In homes throughout the lower income nations, the most common flame burn risk is associated with kerosene lamps and stoves. These two appliances are in constant use for cooking and lighting. Burns occur when these appliances malfunction and kerosene sprays out while people attempt to clean and fix them. One study from India reported that a kerosene stove was involved in 80 percent of the flame burn injuries treated at a burn unit. Customary cooking at floor level was cited as an additional risk factor. Flame burns would be reduced dramatically worldwide if people used efficient, safe, inexpensive kerosene lamps and stoves placed above floor level. Keep children out of kitchen. Avoid storing cooking material across the stove. Avoid bending over the flame to put out fire. Avoid keeping stove near gas cylinder.[39]

Flammable Fabrics

In many higher income countries, clothing ignition burns have become quite rare and those which do occur are often linked with flammable liquids. Reasons for this reduction include: flammability standards for children's nightwear, a trend toward closer-fitting garments for women and girls and the redesign of ignition sources, such as space heaters. However, clothing ignition is a major problem in countries where women's traditional dress is loosely draped (saris) and made of cotton or rayon. The beauty of these garments and pride in the traditional dress has made it unlikely that reductions seen elsewhere will occur. These garments combined with floor-level cooking, place women at very high risk. Research is needed in the development of inexpensive flame-resistant materials that can be used in the manufacture of saris and other traditional women's clothing. While cooking, the women should be encouraged to wear only those designs of traditional dress which fit closer to the body and are thus, less likely to catch fire at the stove. Avoid using 'sari' or 'dupatta' to handle hot vessels.[39]

Cigarette

Lighted tobacco products, primarily cigarettes, are the major cause of fire deaths in higher income countries, for example, approximately 50%in Sweden,[40] 33% in Canada and 25% in USA.[41] In Japan, smoking materials cause 14% of fire-deaths, second only to incendiary suicide (23 %).[42] In the USA, the Center for Fire Research at the National Institute for Standards and Technology has developed a test method to measure compliance with a performance standard to reduce cigarette ignition propensity. Once this standard is adopted, death and injury from cigarette-ignited fires should drop dramatically around the world. It is necessary that all cigarettes manufactured, imported or exported, and/or sold within a country meet the performance standard for reduced cigarette ignition propensity. Home owners must install smoke detectors in all owner-occupied and rented homes. Educate people not to smoke in bed or under influence of alcohol and drugs. Extinguish all cigarettes before throwing. Redesign cigarette to make them fire safe. Ban use of cigarette at places like petrol pump.

Hot Liquids

Do not keep hot liquids/foods near the edge of table. Keep them out of the reach of children. The temperature of the hot water heater should be set at 124° F. Check temperature of water before bathing children. Never leave a child unattended in the bathroom. Avoid leaving unattended hot liquids. Do not use loose pressure cooker rubber (casket). Do not carry hot water on a slippery floor. Avoid carrying hot liquids when children are around. Do not pour water in an empty hot car radiator, wait to cool or keep the face away from the radiator while pouring water.[42]

Liquid Petroleum Gas (LPG) Leak

Windows and door should be kept open. Do not light a match or switch on the electric light. Call Gas Company or Fire Department.[42]

Fire Works/Fire Crackers

Fire crackers should be ignited from a distance and those which fail to burst should not be inspected from close distance because it can burst at any time. Do not wear easily flammable fabrics while playing with fire crackers. Do not leave children to play with firecrackers unsupervised. Redesign fire crackers with safety measures. Ban dangerous fire crackers. Use fire works in the open ground only.[42]

Crowded Places (Theater)

Heat and smoke detectors should be installed. A well designed fire escape plan should be available. Fire brigade and police telephone numbers should be kept near phone. Fire extinguishers, water and sand should be available readily.[42]

Electric Burn

Educate children not to touch wires and electrical appliances. Do not handle electric appliances with wet hands and bare feet. Unplug electric appliances when not in use. Use standard company electric appliances. Plug point should be placed at higher level out of the reach of children and it should be covered with a cap. There should be no dangling cords. All flawed cords should be replaced with redesigned cords. Keep electric appliances away from the water. Hot iron should not be left unattended within reach of children. Avoid open plug point. Avoid open bathrooms if immersion rod is used to heat water. The rescuer must be careful so that he does not become a part of the electrical circuit in attempting to free a person still in contact with a live wire. Use dry wood and push the patient away from current source.

When flying a kite, it is important to remember the 3 C's of kite safety: Caution, Courtesy and Common Sense. By keeping these three items in mind at all times, you will make your recreational time more enjoyable, and safer[43].

Here are some extra tips for making your kite outing a safe one:

- Choose a flying site at least 3 miles (5 km) away from airfields; also stay away from roads and railway lines.
- Never fly your kite near electrical pylons, overhead power cables or overhead lines of any kind.
- If your kite becomes entangled, do not attempt to free it yourself. Seek help from your local authorities.
- Do not fly your kite in electrical storms. The kite will act as a lightning conductor, causing injury and even death.
- Be aware of casual observers. They could be unaware of the potential dangers involved in kite flying.
- Flying kites on larger lines can cause line burns on your hands, so be sure to wear gloves.
- Always be aware of who or what is behind you as well as in front of you.

- Know your skill level, limitations and strength. Do not fly anything too large for the conditions or try any complicated moves in crowded areas.[44]

SUMMARY

Burn injuries in a developing nation like India are still a major problem. Poverty, illiteracy, lack of funds and lack of burn education, etc. are the major hurdles in solving the problem of burns in India. The only answer is Burn Prevention. It is not quick, cheap or easy. If it were, the burn problem would have been eliminated long ago. Be prepared to make long-term plans to gather public support for safer product designs and for codes and regulations which are often necessary for effective burn prevention. Be prepared to rethink strategies halfway through a project. Be prepared for a struggle. But know that burns are preventable.

A final word: 'Prevention takes time, energy and money but ultimately, it is the solution to the world's burn problem'. 'Vision is nothing but a dream that's backed by the drive to achieve it'.

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Chapter

2

Anatomy of the Skin

Sujata Sarabahi

INTRODUCTION

Skin is the largest organ, covering a surface area of 1.5 to 2.0 m^2 in an adult. In burns, although there is a direct damage by the causative agent to this outer protective layer of the body, almost all the other organ systems are indirectly involved, in addition. Before considering the pathophysiology of burn wound, it is imperative to understand the anatomy and physiology of normal skin as both are disrupted following burns.

The skin consists of a thin outer layer which is cellular and derived from the ectoderm – the epidermis and an inner thicker fibrous layer which originates from the mesoderm –the dermis (Figs 2.1 and 2.2). The two layers are separated by a basement membrane or basal lamina. Nerve endings are present throughout both the layers. The connective tissue of the dermis provides a firm structural base for the skin. Underneath the skin lie the subcutaneous tissues, muscle and bone. The thickness of skin is variable in different anatomical areas of the body, in different sexes and at different ages in the same individual. This produces differing depths of burn injury in different parts of the body and at different ages from a given temperature and time of exposure. In general, the skin of infants, children and elderly is thinner as compared to that of adults. Thus, the burns may be deeper in children and elderly with the same agent causing a comparatively superficial injury in adults. Males have thicker skin as compared to females.

The skin of the scalp, palm and soles is thickest (2 mm thick) compared to the rest of the body, whereas it is thinnest on the eyelids (0.5 mm thick) and external genitalia. This is because of the difference in degree of keratinization of the epidermis. The degree of keratinization of skin is largely under genetic control. However, intermittent mechanical pressure or abrasion stimulates adventitious production of keratinized cells which enhances stress resisting properties of skin. The interface between epidermis and dermis is marked by peg and socket or ridge and groove interdigitations which along with specialized anchoring structures linking epidermis to dermis prevent the epithelium from being stripped off the surface of dermis by shearing forces.

ANATOMY OF EPIDERMIS

Normal epidermis is a terminally differentiated, stratified squamous epithelium divided into five distinct layers each of which represents different stages in the development of the fully mature cells of the outermost layer—the **Stratum Corneum** (as we go from the innermost layer to the outermost layer). The thickness of stratum corneum varies in different parts of the body and it is subjected to constant shedding. It consists of several rows of cornified, non-nucleated dead cells containing keratin. This layer forms a barrier on the surface of the skin, protecting the underlying living cells from damage by trauma and desiccation.

The **Stratum lucidum** lies immediately below stratum corneum and is found only in the skin of soles of feet and palm of hand as a thin eosinophilic band. The **Stratum granulosum** is the next layer of flattened nucleated cells containing a precursor of keratin called keratohyaline granules. The fourth layer is the **Stratum spinosum** which is prickle cell layer containing several layers of polyhedric and cylindrical cells.

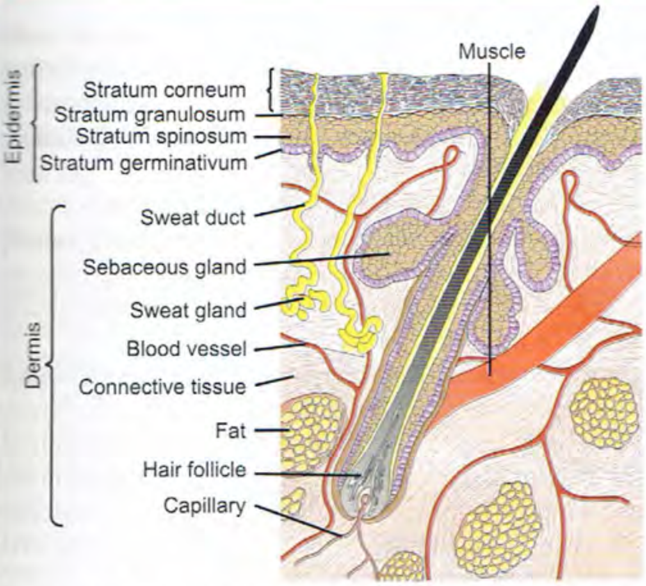


Fig. 2.1: Diagrammatic representation of anatomy of skin

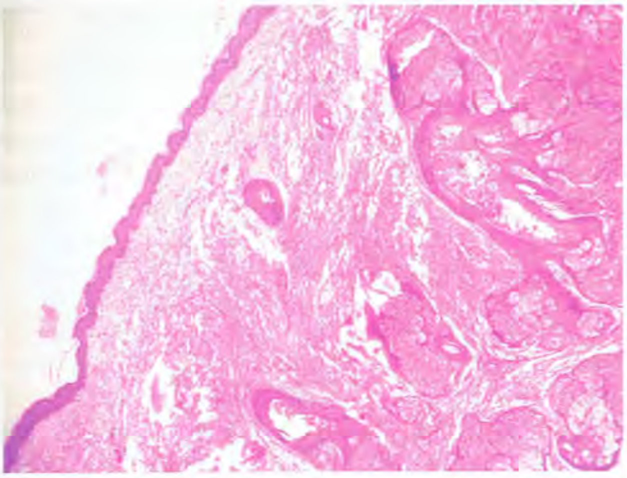


Fig. 2.2: Normal skin on histopathological examination (*Photo courtesy: Dr Geetika Khanna*)

The deepest layer is the **Stratum Basale** or the germinal layer of the epidermis. This consists of a single layer of pigment granules containing cuboidal or columnar shaped cells called keratinocytes which are in a state of high mitotic activity. These epidermal stem cells in the basal layer divide to form transient amplifying cells which then migrate suprabasally, stop dividing and undergo terminal differentiation. This process of maturation and migration from basal layer to outermost layer takes about 26-42 days. The keratinocytes are attached to each other by desmosomes which can be broken and reformed allowing the mature keratinocytes to migrate upwards. The desmosomes of the stratum corneum are also degraded before desquamation so that there is shedding of outer layers of epidermis as deeper layers move outwards. The time taken for the deepest layer of stratum corneum to reach the outermost layer and desquamate is approximately 14 days. Thus, the normal epidermis completely replaces itself in 45-75 days.

Within the germinative layer of epidermis, there are three other cell types, viz., the melanocytes, the Langerhans cells and the Merkel's cells.

Melanocytes are dendritic cells which produce pigment granules called melanosomes which contain melanin pigment. Majority of these melanocytes are located at the dermo-epidermal junction beneath the basal cells and some lie in the dermis. Embryonically, melanocytes are derived from the neural crest and they migrate to the epidermis. The dendritic processes of the melanocytes contain the melanosomes and once their tips are sheared off, they are phagocytosed by the keratinocytes. Thus, the pigment is transferred from the melanocytes to the keratinocytes.

The rate of melanin production, transfer to keratinocytes and degradation of melanosomes determine the degree of skin pigmentation. The ratio of melanocytes to epidermal cells is 1 in 5 on face and 1 in 20 in the buttocks. The average ratio of melanocyte to keratinocyte on the body varies from 1:4 to 1:10.The relative number of melanocytes is approximately equal for men and women and for all races. The difference in color between races is not due to difference in number of melanocytes but due to activity, rate and production of melanin. For example, individuals with darker skin have very large melanosomes which are fully melanized as compared to individuals with fairer skin who have smaller and less melanized melanosomes. This is to be kept in mind when choosing a donor area for procuring the skin graft. There will be a definite difference in color when skin from an area of higher melanosomes content is grafted on to areas that have a smaller number of melanosomes.

The melanin protects epidermis from harmful effects of ultraviolet rays of Sun by forming a protective layer around the nucleus of the keratinocyte so that these rays do not damage the DNA in the nucleus. A higher amount of melanin, thus, protects the skin from Sun burn which is a first degree epidermal burn of skin on increased exposure to sunlight.

Langerhans cells are also dendritic cells found in the epidermis but devoid of melanin. They contain rod like Langerhans granules, some of which are in continuity with the external membrane of the cell. Their function is still unclear. Recent research has shown that these cells are a type of phagocytic cells and they provide a 'trap' for various contact antigens and present them to T–cells for destruction. Thus, they protect the internal structures from the potentially infectious agents in the external environment.

Merkel cells are found in the basal part of epidermis of hairy skin. They are in close contact with the tactile sensory nerve endings terminating in the basal part of the epidermis. Thus, they play an important role in the sensory processes.

The basement membrane at the dermo-epidermal junction is a highly organized structure of protein that anchors the epidermis to the dermis. It consists of grooves on the epidermal side called epidermal ridges which fit into projections from the dermis called dermal papillae. A number of microfilaments insert into this basement membrane and get aggregated in the adjacent papillary dermis resulting in structures called 'rete pegs' or 'rete ridges' which are seen as invaginations into the papillary dermis to increase the surface area. These resist the shearing forces between epidermis and dermis. Any gap due to shearing between epidermis and dermis can produce a blister as is seen following superficial dermal burns.

ANATOMY OF EPIDERMAL APPENDAGES

The functions of human skin are further affected by certain cutaneous appendages which are of ectodermal origin. These are the hair follicles, sebaceous glands, apocrine glands and sweat glands.

Hair Follicle

Hair are found on nearly all parts of the human body except palm of hands and sole of feet, glans penis, inner surface of prepuce and clitoris, labia majora and minora. The hair has a root which is the part inside the skin and a shaft which is projecting outside the skin. The root of the hair has a proximal enlargement, the hair bulb, which is set in an invagination of the epidermis and superficial portion of the dermis called the hair follicle. The hair follicle commences on the skin surface with a funnel shaped opening and then passes obliquely downwards to become dilated at its end which corresponds to the hair bulb. The ducts of one or more sebaceous glands open into the follicle near the skin surface. In the hair bulb there is a small conical vascular prominence or papilla which is continuous with the dermal layer of the follicle and is supplied with myelinated and nonmyelinated nerve endings. The nutrition to the hair is derived from the capillaries in this papilla.

The hair follicle has two coats—an outer coat which is mainly fibrous tissue and is continuous with the dermis and an inner coat or root sheath which is closely adherent to the root of the hair and is basically a continuation of the epidermal cell layer. Since the hair follicle is primarily composed of epidermal tissue, it has a significant role in re-epithelialization of burn wounds.

The hair bulb is moulded over the papilla and consists of polyhedral cells which are undifferentiated and rapidly dividing. The products of these rapidly dividing cells are the inner root sheath and hair which move outwards towards surface of the skin.

The hair shaft is a dead cornified structure which extends from the follicle above the skin surface. The shaft consists from within outwards, the medulla, the cortex and the cuticle. The color of the hair depends on the amount of melanin and its distribution in the cortex. Blond hair has few melanosomes while gray hair does not have melanosomes due to a relative absence of melanocytes.

The terminal hair follicle has three phases in its growth cycle, the active phase or anagen, the involuting phase or catagen and the resting phase or telogen. The hair follicles produce hair independent of each other.

In burns, unless the wound is very deep or full thickness, the epidermal tissue lining the hair follicles is still available in the depths of the dermis in the wound site to generate new epithelium. The hair also traps air and provide insulation, though this is true only for the scalp.

Sebaceous Glands

These are small sacculated glands arising from an epithelial bud from outer hair root sheath. They are found in most parts of the skin, more abundant in the scalp and face while being absent in the palms of hand and sole of feet. Each gland has a single duct which emerges from a cluster of oval or piriform alveoli. Each alveolus has a basement membrane enclosing a number of epithelial cells. The marginal cells are small and polyhedral and in continuation with the cells lining the ducts. The remainder of the alveolus is filled with larger cells containing fat. In the center, the cells are broken up leaving a cavity filled with a mass of fatty matter called sebum cutaneum. Since the sebaceous glands produce their secretions by complete fatty degeneration of their central cells they are classified as holocrine glands. Commonly, their ducts open into the hair follicles. Occasionally they open on to the general surface. Sebum acts as a natural lubricant for the hair and skin and thus protects them from desiccation. It has bactericidal action as well. Their secretory activity is under hormonal control, especially androgens. Following a burn injury, if the sebaceous glands are damaged or the raw area is split skin grafted the new skin may not be able to secrete enough sebum to lubricate the skin. Hence, a frequent application of moisturizing creams/bland oil is required to prevent the dried skin from cracking and subsequent infection.

Sweat Glands

Sweat glands are present in the skin of almost every part of the body and are classified into two types: eccrine and apocrine.

The eccrine glands are more numerous. They consist of a single tube which is coiled into a spherical or oval ball called body in its deep part which is situated in the deeper layers of the dermis or in subcutaneous tissues. The duct traverses the dermis and epidermis and opens on to the surface of skin by an aperture. The size and number of glands vary in different parts of the body. They are large in axilla and groin where the amount of perspiration is more. They are also numerous in palm and sole. The tube consists of two layers –an outer layer of fine areolar tissue which is continuous with superficial layer of dermis and inner epithelial layer consisting of cubical or polyhedral cells. Between the basal aspects of these cells and basement membrane, there is an incomplete layer of myoepithelial cells which help in the expression of secretions from the gland by virtue of their contractile property. The ducts are composed of a basement membrane lined by two to three layers of polyhedral cells. These eccrine sweat glands are merocrine in nature, i.e. they produce their thin watery secretions without epithelial cell disintegration.

Apocrine sweat glands are present in the axilla, eyelids, nipple, areola and circumareolar region of the breast and in the external genitalia. They are in close association with hair follicles and produce thick secretions. They contain a coiled secretory gland in the lower dermis and a straight excretory duct. Their secretions are sterile and odorless within the duct but once excreted onto the surface of skin, bacteria degrade them resulting in body odor.

The primary stimulus for the production of sweat is heat. The thermoregulatory center in hypothalamus is responsible for sweat production in an effort to maintain body temperature as sweating helps in dissipating heat. A patient who has sustained deep dermal or full thickness burns looses majority or all of his eccrine sweat glands and if significant body surface area is involved in these burns, the patient's rate of sweating becomes inefficient. Thus, these patients may not be able to regulate their body temperature by sweating in hot and humid surroundings leading to a rise in their core body temperature.

ANATOMY OF DERMIS

Dermis is also called Corium. It is a tough, flexible and highly elastic structure. It is very thick in palm of hand (1.6 mm) and sole of feet and very thin in eyelids (0.4 mm), scrotum and penis. It consists of mainly connective tissue with varying number of elastic and collagen fibers, numerous blood vessels, lymphatic vessels and nerves. The fibroblasts are the 'master cells' of the dermis which produce the collagen fibers, elastic fibers and ground substance. Collagen forms approximately 77% of dry weight of dermis and provides tensile strength to it. Eighty five percent of the collagen is Type I and 15% is Type II collagen. Elastic fibers are interwoven within the network of collagen fibers. The connective tissue in dermis is arranged in two layers, superficial papillary dermis and deeper reticular dermis.

Papillary dermis consists of numerous papillae which are highly sensitive vascular conical projections which project perpendicularly into corresponding pits on undersurface of the epidermis. Each papilla consists of small closely interlacing bundles of fine fibrillar tissue with a few elastic fibers and within this there is a capillary loop. In some papillae, especially on the palms and fingers, there are tactile corpuscles. The papillary dermis primarily consists of small loosely distributed elastic and collagen fibrils with a greater proportion of interfibrillar gel and connective tissue that encloses the microcirculation and lymphatic network below the papillary projections.

Reticular dermis is the thicker part of dermis and is more acellular and avascular compared to papillary dermis. It consists of strong interlacing bands of white fibrous tissue and some yellow elastic fibers. The collagen bundles are arranged mainly parallel to the skin surface and some are perpendicularly arranged forming a lattice work. This arrangement of collagen fibers in reticular dermis provides skin with extensibility in different directions. The directions taken by the parallel bundles of collagen fibers form the cleavage lines of the skin. Surgical incisions given along the direction of cleavage lines heal with minimal scar tissue whereas incisions perpendicular to these lines cause retraction of the severed fibers, leading to a broad scar. With increasing age, the yellow elastic fibers atrophy and skin loses its elasticity and becomes wrinkled.

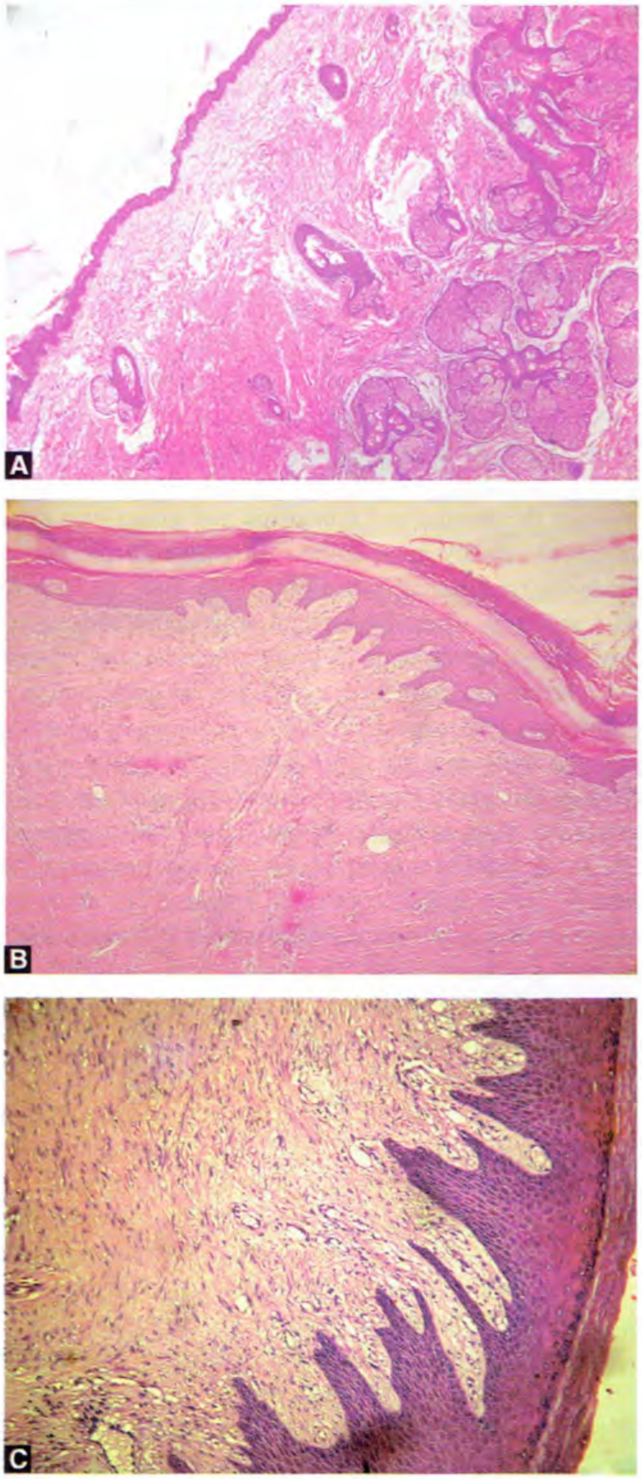
Following burns if only the epidermis is involved, they heal without any scarring of skin. However as the deeper layers of skin are involved, depending on depth of dermis involved, the skin always heals with scarring. Again, depending on amount of collagen production and the arrangement of collagen fibers there can either be hypertrophic scarring or keloid formation (Figs 2.3A to C). Details of the pathophysiology of post burn scar formation has been described in detail in the Chapter On Wound Healing in Burns.

Deep to the reticular layer is the subcutaneous areolar tissue which contains fat. In many regions, the skin is separated from the deep fascia or other structures by loose areolar tissue and in these sites the skin is freely movable over the deeper structures. Elsewhere, however, the skin may be firmly anchored to structures like the periosteum over subcutaneous parts of bones or to deep fascia in regions related to movements of underlying joints where it leads to permanent creases in the skin known as flexure lines, e.g. palm of hand and flexure surface of digits.

BLOOD SUPPLY OF SKIN

The blood supply and drainage of skin lies along well determined pathways which depend upon the metabolic requirement of various cellular components. The most active areas of the skin are the epidermis and its appendages and the dermal papillae which contain active fibroblasts and sensory endings of cutaneous nerves. The reticular layer is highly fibrous and contains few active cells; therefore, its metabolic requirement is low. There are two vascular plexuses –one at the dermal-subcutaneous junction called rete cutaneum and one in the papillary dermis called rete subpapillare or superficial plexus. These are connected by vertical vascular channels. In the papillary plexus there is a network of arterioles, capillaries and venules which provide a large amount of blood supply to this layer. Capillaries extend from this layer towards the base of epidermis within the dermal papillae and loop back to flat venous plexus which lies immediately beneath the rete subpapillare which drains into venous plexus at dermis-subcutaneous junction via another intermediate plexus in the middle of reticular dermis. Various capillaries in dermis around glands and hair follicle also drain into these three venous plexuses at different levels in the skin.

Apart from providing nutrition to the skin, the cutaneous vasculature has an important function of thermoregulation. Another important role of microcirculation is in the inflammatory process seen in the skin following any kind of trauma which helps in healing process. Following burns involving only superficial layers of skin, the microvascular



Figs 2.3A to C: (A) Mature scar with atrophy of overlying epidermis and bundles of fibrocollagen in the dermis. (B) Histopathology of Hypertrophic scar (H & E, 100 X) showing increased keratin, epidermal hyperplasia with marked increase in collagen in the upper dermis. (C) Higher magnification of a Hypertrophic scar (H & E, 200 X) showing marked epidermal hyperplasia with collagenization of the upper dermis (*Photo courtesy: Dr Geetika Khanna*)

circulation in the deeper layer is intact. Therefore, the deeper vascular bed of the skin has the potential to heal rapidly as it promotes the re-epithelialization process.

LYMPHATIC DRAINAGE OF SKIN

The lymphatic drainage of skin is via numerous blindly ending lymphatic vessels which terminate in the dermis near the base of epidermis and drain first into a dermal network in papillary layer and then into another network in the middle of reticular layer and finally into a network at the junction of dermis and subcutaneous tissues. Deeper to this, there are wide channels with valves which drain into the main lymphatics of the area.

INNERVATION OF SKIN

Skin is richly supplied by myelinated and nonmyelinated sensory fibers of craniospinal nerves and via nonmyelinated autonomic fibers which supply blood vessels, sweat glands and hair follicles. The nerves penetrate superficial fascia and ramify through reticular and papillary layer of dermis forming nerve plexuses around hair follicles in both the reticular dermis and the papillary dermis. Most of the nerve fibers end in the dermis and some penetrate into basement membrane.

The skin contains sensory organs for all the principle sensations like touch, pain, temperature, pressure, itch and tickle. There are mainly six types of sensory receptors in the skin:

1. Free terminals or free nerve endings form majority of the sensory receptors found in epidermis and dermis. They are related to sensations of itch and pain and fine discrimination for sensory stimuli in digits. As the name suggests, they contain one or more axons enclosed in an extension of Schwann cell which is then covered with a basal lamina.
2. Merkel cells are special cells found in the epidermal ridges having attachments next to keratinocytes. They act as mechanoreceptors for sensation of touch.
3. Meissner's corpuscles are found mainly in the papillary dermis of palm and sole, responsible for sensation of touch and relative pressure associated with touch.
4. Pacinian corpuscles are present in the deep layers of dermis in palm and palmar subcutaneous tissue and near periosteum of proximal phalanges. They are responsible for sensation of vibration and signal for local changes of blood flow.
5. Pilo-ruffini corpuscles are sensory receptors which encircle the hair follicles and therefore, they respond to changes in pressure on skin sensed by movements of hair follicles. They are also responsible for sensation of warmth.
6. Krause's end bulb's are widely distributed in superficial layers of dermis and are end organs for cold sensation. Patient sustaining burns may lose some or all of these sensory receptors depending on depth of burns. As a result, they may lose some of the sensations in the affected areas.

FUNCTIONS OF SKIN

- The most important function of skin is its ability to act as a physical barrier preventing bacteria and other organisms from entering it. It also serves as a water barrier and thus maintains fluid balance in the body. Following burns, the integrity of skin is disrupted allowing loss of fluids from the body and entry of organisms into the body. Thus, hypovolemia and infection are the two major complications to be tackled by a burn surgeon.
- Skin also regulates heat exchange and helps body temperature homeostasis. When the cutaneous vessels dilate the blood flow to skin is increased, as also the amount of heat transmitted to the body surface. When cutaneous vessels constrict, blood flow to the skin is decreased with consequential decrease in heat loss from body. In this way, the body temperature is maintained.
- Similarly, perspiration from the skin surface also controls the body temperature. The rate of evaporative water loss from the burned skin surface is directly proportional to the extent and depth of burns. This evaporative water loss enhances the heat loss from the body as well. Approximately 580 Kcal are required for evaporation of one liter of water from the body surface. The excessive heat loss from the body surface of a burn victim causes a severe metabolic drain as well because this is accompanied by hypermetabolism. This evaporative water loss can be estimated by the following formula:
- Evaporative water loss (ml) = (25 + %TBSA burn) × BSA m^2

  TBSA —Total Body Surface Area

  BSA—Body Surface Area
- The stratum corneum of the skin has the ability for selective permeability allowing absorption of some substances and acting as a barrier to others, while hair follicles and eccrine glands only allow percutaneous absorption. A concentration gradient is required to allow the percutaneous absorption of substances across stratum corneum. This is also affected by the relative hydration of stratum corneum. Lipid and lipid soluble

substances pass through the skin easily compared to hydrophilic organic compounds. A gaseous compound like carbon monoxide can also get absorbed by the skin when present in high concentrations.

- Through its various sensory receptors, the skin transmits sensory feedback from the environment.
- Last but not the least, the skin has a sophisticated immune system which plays an important role in host defenses. Whenever skin is exposed to a new antigen, activated antigen expressing Langerhans cells migrate from the skin to lymph nodes, where they activate naïve T-cells which then become memory T-cells and express new surface markers that allow them to accumulate preferentially in the skin in response to cutaneous injury.

SUMMARY

Knowledge of detailed anatomy and physiology of skin is a must for the better understanding of the mechanisms of alterations produced in burns, the management of burn patient as a whole and the causation and minimization of scars.

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Chapter

3

Burns: Etiology and Classification

Sameek Bhattacharya

INTRODUCTION

Since the discovery of fire, man has been on a relentless quest for energy. This pursuit is driven by the expansion and progress of human civilization. The benefits of fire reaped by man have paralleled the fatalities and disabilities caused by it. The magnitude of this devastating injury has increased with the ability to harness higher energy source, use of fire in most activities of life and its application as a destructive modality. Hence, the epidemiological profile of burn injury has changed with time and advancement of the society. The incidence of burn injury directly reflects the imbalance between the use of fire and the level of fire safety consciousness of a society. Therefore, any preventive strategy must be preceded by adequate and reliable data collection about the cause and circumstances of injury. Burns are classified on the basis of etiology, depth and percentage of body surface area involved.

CLASSIFICATION ACCORDING TO ETIOLOGY

Burn injury is a syndrome of local and systemic manifestations of exposure to very high temperature, electrical energy, chemical and radiation.

Thermal Burns

The commonest cause of burns is thermal, caused by flame, blast, contact with hot objects and hot liquids. Thermal damage to skin is primarily a function of energy transfer from heat source to the body. The heat transmission depends on the temperature of the source, conductivity of the tissues, insulation such as hair and keratin layer and the clothing. The thermal threshold of human skin is about 70°C.

Flame Burns

Flame burns account for over 80% of the total burn cases in India and other developing countries[1] (Fig. 3.1). This is at variance with the western data where scalds are the predominant cause. More than 85% of the flame burns in our country are attributed to accidents of which mishaps in the kitchen account for over 60%.[1] In contrast, space heating and house fires are the predominant causes in the west.[2] The high incidence of cooking accidents is ascribed to unsafe heating appliances, floor level cooking and wearing of loose fitting clothes. The major killer among these is the kerosene pressure stoves, which work on aerosolized kerosene oil (Fig. 3.2A). In the event of carbon-choked burner holes, vigorous pumping of the stove suddenly opens up the holes expressing out a large volume of kerosene aerosol which

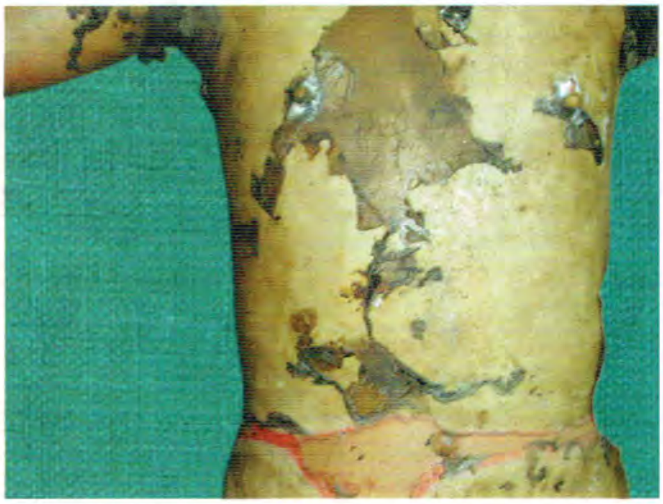


Fig. 3.1: Flame burns leading to extensive injury

ignites into fireball (Fig. 3.2B). This event is frequently labeled as "stove burst". A better version of the kerosene stove is 'wick'stove which works by capillary action (Fig. 3.2C). Another frequent mishap in kitchen is LPG (Liquid Petroleum Gas) leak. The leak can be from a cracked tubing, faulty regulator or from an un-ignited burner. The LPG gas, being heavier than air, does not diffuse out of the ventilators and instead gets settled over the floor. In the event of an ignition, be it lighting of the burner or even switching on of the electric light or exhaust fan, the expressed gas bursts into flames. Although LPG stoves are labeled as safer appliances than kerosene pump stoves, LPG mishaps generally involve a larger number of casualties compared to the latter where only the operator gets burnt (Fig. 3.3). In addition to the inherent unsafe nature of these devices, lack of basic safety consciousness is also to be blamed in these accidents. A frequent practice among the underprivileged class is floor level cooking. The loose fitting clothes of the housewife catch fire when she stands up near the stove to pick up some thing from the overhead kitchen shelves. Another frequent cause of domestic fire accidents is home-made kerosene oil lamps to lighten the house in absence of electricity. These are kept on overhead locations, get tripped by domestic pets, igniting the spilled kerosene oil on unsuspecting victims below.

Homicidal burns account for about 10% of all flame burns.[1] The common mode of this aggression is to douse the victim with kerosene and then set on fire. This frequently results in extensive and deep burns as the victim is generally incapacitated or restrained. Suicidal burns, around 5% of all flame burns, frequently involve kerosene doused over the body by the individual herself/himself in a fit of anger and then set on fire.[1]

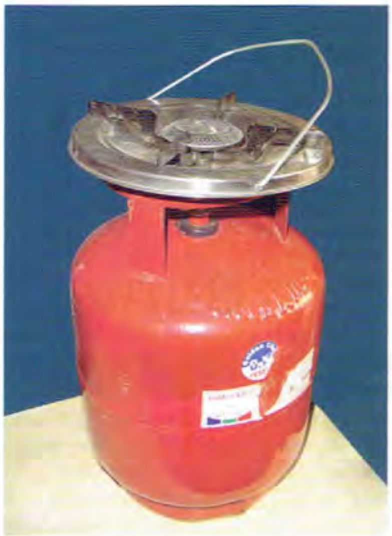
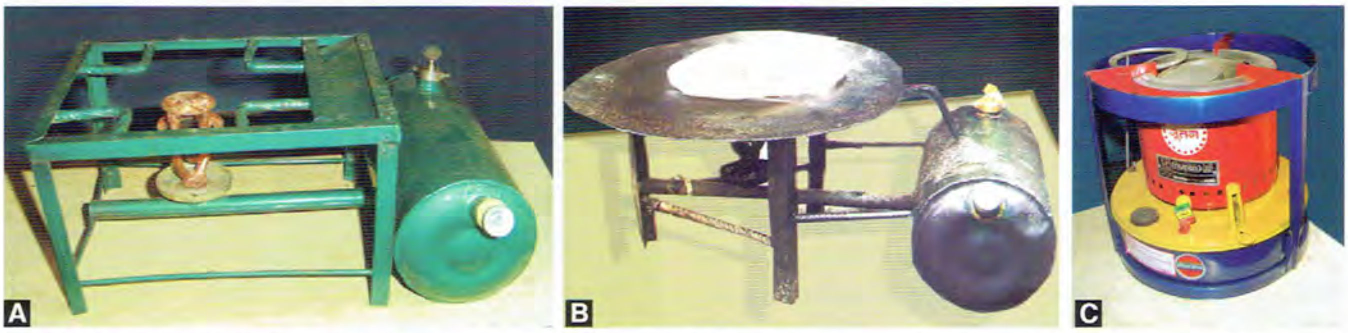


Fig. 3.3: LPG stove

Flame burns on the whole are more frequent in the age group of 16 to 55 years. Also, there is a definite female predominance among flame burns which is more so in the productive age group of 16 to 35 years. This is obvious as traditionally females are engaged in the unsafe kitchens. Homicidal and suicidal burns are also more frequent among females.

Scald Burns

Scalds or burns due to hot liquids account for more than 50% of all burn admission in the developed world [2](Fig. 3.4). In contrast scalds constitute only 15% of all burn admissions in India.[1] In both scenario scalds are more frequent in children. In the developed world, the incidence of flame burns is far less compared to the developing countries because of better safety features in the kitchens.



Figs 3.2A to C: (A) Kerosene pump stove. (B) Kerosene pump stove in action –it is a pressure stove in which continuous pumping aerosolizes the kerosene. If the burner holes are choked and suddenly open up by vigorous pumping of the stove, there is a release of large volume of kerosene aerosol which ignites into a fireball when the stove burner is lit.This is wrongly labeled as 'Stove burst'. (C) Kerosene 'wick' stove—Safer than pump stove

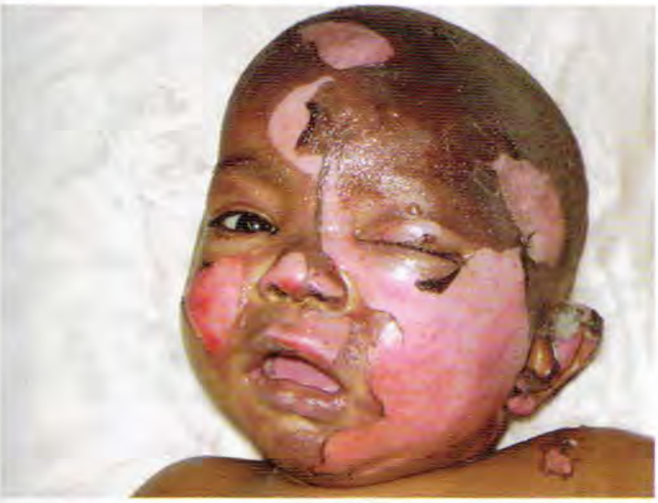


Fig. 3.4: Scalds over face of an infant due to accidental spilling of hot tea

In contrast, incidence of scalds remains high in the west as children are less protected with working parents in nuclear families. In developing countries, on the other hand, children are relatively better off with non-working mothers and in joint families. The incidence of scalds peaks around the age group of 3 to 5 years. This is the time when the child is highly mobile, inquisitive, lacks awareness regarding hazards and devoid of experiences to cope up with them. Most of the accidents take place when the child is unattended or parents are momentarily distracted or engaged with hot liquid with the child in their arms. The extent of scald burns depends on the temperature, volume and density of the liquid, age of the child and the absorbing ability of the garment. A small amount of boiling water causes a higher % age TBSA burn in a small child compared to an adult. Also hot water absorbed by the clothes worn spreads and involves a larger area and causes deeper burns as the exposure time is longer. The sticky sugar syrup, oil or 'ghee' and thick gravy cause deeper burns as they can be extremely hot and stick to the skin. Due to their thin skin, children suffer full thickness scald burns more frequently than adults. Commonest circumstances of scalds in developed world are during bath followed by kettle. On the contrary, Indian children get scalded while the mother is handling hot food or water with child in her lap or around her. Children also get burnt when they "reach and pull" containers of hot liquid kept on tables. They either pull table cloth or the container itself. If the container falls over the child, face and upper trunk gets burnt. If the liquid splashes on the floor the legs get burnt. The sitting child gets perineal and thigh burns if it falls on his/her lap. A toddler sometimes tumbles into a large liquid container suffering 100% burn injuries. Overall, the boys get scald injuries more frequently as compared to female children. Although most scalds are accidental, a small definite percentage has been attributed to child abuse in the west. Such cases are a rarity in India. Thermal burns in adults are also mostly accidental. The accidents occur, while preparing tea or gravy or boiling milk and from spillage from hot water bottle. Steam burns are caused by faulty pressure cookers and on forcibly opening car radiators. Scalds and steam burns are also seen in boiler rooms of factories, ships and steam locomotives. Steam having a high latent heat causes severe burns and can cause upper airway edema on inhalation.

Thermal Contact Burn

Thermal contact burns are caused by touching hot objects. Unlike flame and scald burns the injury is always localized to the area of contact. Depending on the temperature and duration of contact the burns can be very deep. In domestic settings, contact thermal burn is caused by touching hot utensils, room heating coils, light bulbs, bike silencers, etc. Contact burns are frequently seen in epileptics who fall on stoves or hot objects during an attack. Due to post-ictal unconsciousness, the prolonged contact may lead to very deep burns up to bone. Contact burn injuries are also seen in insensate hands and feet. Contact thermal burns in factories are caused by hot machine parts, molten metals and coal tar. These burns are also seen in case of torture and commonly used modalities are hot metals, cigarette butts, etc.

Blast Injury

Other than warfare, blast burns happen in abundance during Diwali and other festive occasions. The ill defined legalities regarding sale and use of firecrackers and lack of safety consciousness among people, explain the high incidence of such injuries. Most of the victims are males and are in the age group of 10 to 25 years. The victims are mostly foolhardy "bravehearts" or unsupervised children. Although the firework operator is more frequently injured, about 15% of the victims are bystanders. Most incriminating firecrackers are *'bomb'*, *'anar'*, and igniting of collected firework powder accounting for more than 60% of cases.[3] The injuries are sustained whenever the explosion is either delayed or earlier than expected. Hands and the face are mostly affected. A 'bomb' or an *'anar'* exploding in the hand causes extensive damage and disruption and even loss of digits. An explosion on the face of an inquisitive child stooping over a lighted cracker not only involves the face but also damages the eyes. The ocular injury can range from reactive conjunctivitis, corneal abrasion and perforations. Misdirected rockets accounting over 10% of cases always affect unsuspecting bystanders who get burnt when their clothes catch fire. Firecrackers like '*phooljhari*' and 'zameen chakri' account for minority of injuries which are purely thermal burns[3] (Fig. 3.5).

In addition to the scattered cases, mass burn casualties due to fire in firecracker factories, storage depots and markets are also reported during Diwali. Adjoining residential buildings and parked motor vehicles may also come in range of these fires.

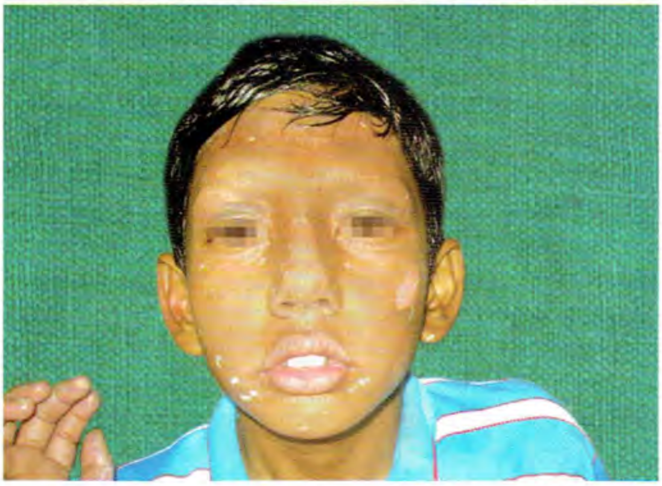


Fig. 3.5: Firecracker injury causing thermal burns of face

Electrical Burn

Electrical burn account for only 5% of the burn cases but are generally extremely devastating.[4] Majority of the cases are males and are in the age group of 10 to 30 years. Electrical injury is broadly divided into low and high tension injury, the demarcating voltage being 1000 volts.[4] The injury is also classified as flash or contact burn depending on the mode of injury (Figs 3.6A and B). Flash burn mimics thermal burn and contact burn is manifested by deeper tissue destruction. The low tension injury is generally seen in domestic settings and is characterized by flash burn on the surface with varying degrees of deeper tissue destruction depending on the site and duration of contact. Such mishaps happen with unprofessional handling and repairing of meter boxes, switchboards and domestic electrical appliances. Although most victims are young males, children get electric burn when they try to mouth or handle a live wire or poke finger in a plug socket (Fig. 3.7).

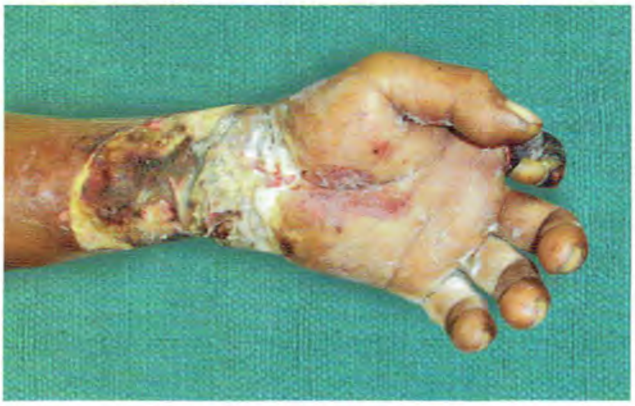


Fig. 3.6A: Electrical contact burn to hand and wrist

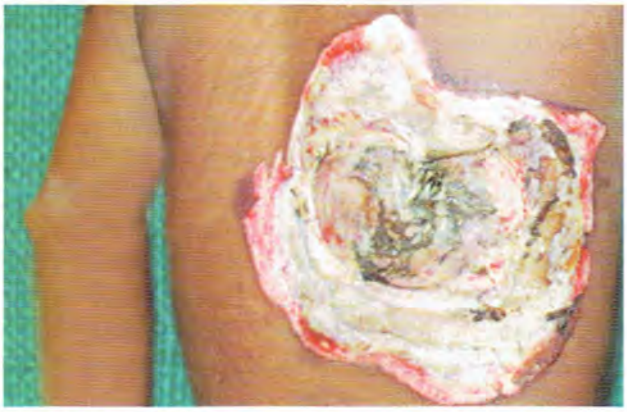


Fig. 3.6B: Electrical contact burn due to contact with high tension wire on back of trunk

High tension burns are commonly seen in linemen working on high tension wires and in factory settings. Such injuries are also seen in cases of attempted illegal tapping of electricity from high tension wires. These cases are on the rise due to mushrooming of unauthorized dwellings near high tension cables (Fig. 3.8). Construction workers in these areas are also frequent victims. High tension injury has also been reported from fallen live cables lying in agricultures fields and from passengers travelling on roof top of electrified trains. Young children are known to get injured while flying kites with the thread getting entangled in high tension cables, especially in rainy season [5](Fig. 3.9).

Chemical Burn

Chemical burn is non-immunological destruction of the skin due to acute exposure to hazardous chemicals. Unlike other forms of burn, there is extensive and deeper damage owing

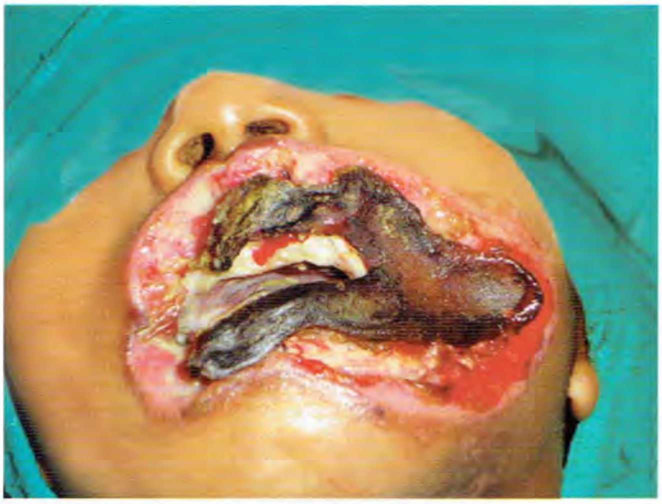


Fig. 3.7: A child with loss of tissues of face due to electrical contact burn by chewing of electrical wire in mouth

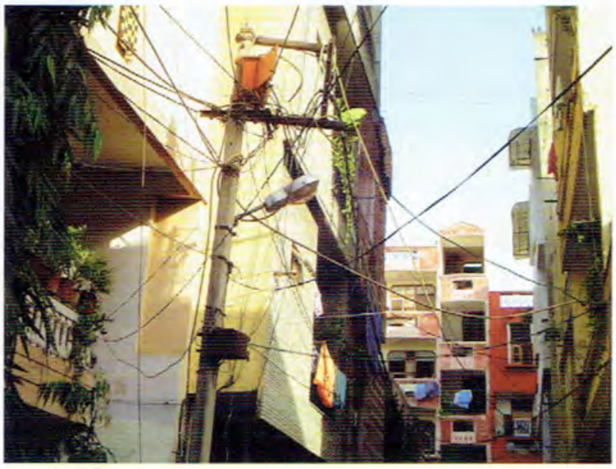


Fig. 3.8: Unauthorized residential areas near electrical pole with wires running close to the balcony of the houses

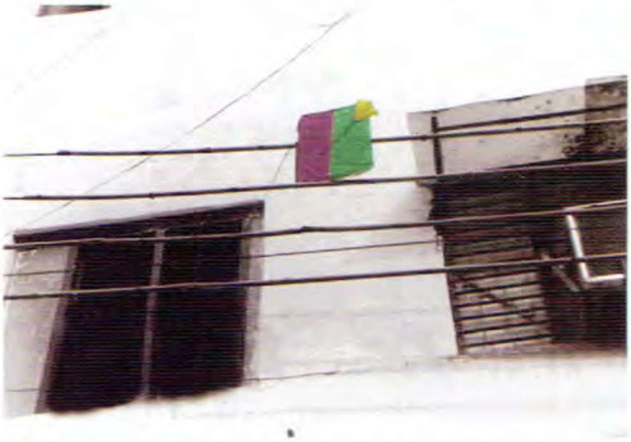


Fig. 3.9: Kite entangled in electric wires – common cause of electrical injury in children during kite flying

to prolonged exposure and continuous reaction of the chemical with tissues. More than 25,000 chemicals are known to cause chemical burns and they are broadly classified according to their chemical nature (Table 3.1).[6] Chemical burns are reported in industrial accidents, civilian mishaps and chemical warfare. The chemicals involved in civilian injuries are acids (40%), inorganic products (30%), alkalis (20%) and organic products (10%).[1] During warfare, the commonest chemical implicated is phosphorus followed poisonous gases like phosgene and Dimethyl sulfoxide (DMSO). Hence, the incidence and pattern of chemical burn varies with countries, societies and situation. Overall incidence of chemical burn is about 2-4% of all burn admissions during peace time. In the industrialized western world majority are reported as accidents from factories handling hazardous chemicals and the victims being male adults of working age. On the contrary, in developing countries like India most of the chemical burns are of non-industrial nature. Most of the domestic accidents are due to mishandling of household acid cleaners which are frequently stored in discarded mineral water bottles and victims are both adults and children. In addition to homes children also get chemical injury while working in school laboratories. Homicidal vitriolage account for a major share of chemical burns in India, while such cases are rare in the west. The majority of victims of this depraved aggression are women who reject the advances or proposals of local goons. The accidental as well as homicidal chemical injuries commonly involve face, upper trunk and upper extremities. Loss of vision due to ocular damage is also frequently reported (Fig. 3.10). The agents responsible for chemical injury are listed according to their mechanism of action in Table 3.2.[1,6]

Table 3.1: Different types of chemical which can cause burns

Chemical Nature	*Agent*
Acids	Hydrochloric acid Nitric acid Hydrofluoric acid Chromic acid Sulfuric acid
Alkalis	Sodium hydroxide Potassium hydroxide Calcium oxide Metallic Sodium Metallic potassium Sodium hypochlorite
Inorganic	Phosphorus Cyanide
Organic	Phenol Lysol Cresol

Radiation Burn

Incidence of radiation injury during peace time is far lower than other types of burn. These mishaps are reported from the thermonuclear explosions, nuclear reactor accidents, radiography industries and X-ray departments. Burn injury from non-ionizing radiations like radio and micro waves are rare and minimal as they have minimal tissue penetration. Minor burns are reported with prolonged exposure to microwaves. On the contrary ionizing radiation like gamma rays, X-rays and high energy particles like protons penetrates deeply into tissues causing serious burn injuries. There has been around 50 radiation accidents reported till date other than Three Mile Island and Chernobyl nuclear reactor mishaps and Hiroshima Nagasaki bombings.[1,7]

Laser Burns

Burns caused by lasers are basically thermal burns where laser energy is used for other purposes like hair removal or resurfacing of vascular or pigmented lesions and heat released is dissipated into the normal skin leading to skin burns. The damage to the skin will depend upon the energy released by the laser. Therefore, these can be superficial burns which are more common or deep dermal, when the energy used is very high ,which take a long time to heal. Fires also have been reported with use of lasers whenever there are flammable substances present in the operating field like oxygen used during anesthesia, dry gauze, alcohol, perfume, nail polish, etc. Hence fire extinguishers should always be available near by (Fig. 3.11).

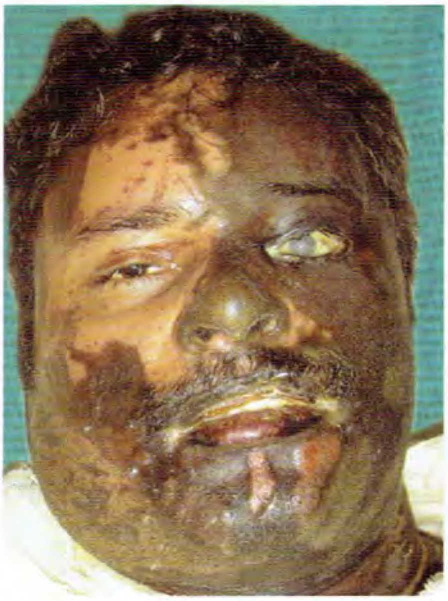


Fig. 3.10: Chemical burns of face involving eye

Friction Burns

Burns due to friction are one of the commonest non thermal burns. In majority of the cases they are associated with mechanical injury, which may be more severe than the burns. Usually, friction causes partial thickness burn in a small

Table 3.2: Mechanism of action of various chemical agents

Mechanism of Action	*Agents*	*Use*	*Effect*
Oxidation	Chromic acid	Bleach, Chlorinator	Corrosive type damage
	Sodium hypochlorite	Disinfectant	Does not penetrate deep
	Potassium permanganate		
Dehydration	Sulfuric acid	Toilet cleaner	Coagulative necrosis by cellular desiccation and exothermic reaction
	Hydrochloric acid	Toilet cleaner	
	Oxalic acid	Rust remover	Dry eschar prevents deep penetration
Denaturation	Hydrofluoric acid	Rust remover	Denature and forms salt with vital cell proteins
	Tannic acid	Metal cleaner	
Corrosion	Sodium, potassium and ammonium hydroxide	Drain, Oven cleaner Paint remover	Liquefactive necrosis and saponification by combining with protein and lipids
	Phenol	Disinfectant	Soft burn and deep penetration
Vesicant	Gasoline	Fuel	De-fattening properties produce tissue amines and local tissue anoxia
	Chloro di-ethyl sulfide (Mustard gas)	Chemical warfare	Local irritation, blister and corrosive damage on prolonged exposure
	Methyl bromide	Insecticide	
Self Ignition	White -phosphorus	Military ammunition	Thermal burn, particles get embedded in skin with blast and cause pain and prolonged damage
	Yellow phosphorus	Fire works Flares	

body surface area which is ignored. Whenever there is friction between two objects, like between a body part and a machine or tyre or road, the relatively delicate object, i.e the skin bears the brunt of the mechanical trauma in the form of abrasion, laceration or avulsion. Hence, most often the burn takes a back seat and the mechanical injury becomes the priority (Fig. 3.12).

CLASSIFICATION ACCORDING TO BURN DEPTH

ESTIMATING BURN DEPTH

In addition to etiological categorization, a burn injury is also classified on the basis of depth. Whereas, etiological classification dwells on the epidemiology, classification based on burn depth is purely prognostic. Burn depth determination and its exact categorization are fundamental in electing a conservative or surgical modality of management. Although the prognostic importance of burn depth was postulated by Jean de Vigo in 1483, Peter Lowe in 1597 was first to classify burns into *"superficial, average and great"*. Fabricus in 1610 classified burns into three degrees and was first to recognize erythema, vesication and eschar formation. Later Heister in 1743 and Richter in 1799 recognized four degrees of burn but were highly subjective as pain in addition to erythema eschar formation and vesication. The classical classification of burn depth was propounded by Dupuytren in 1832, which is as follows:

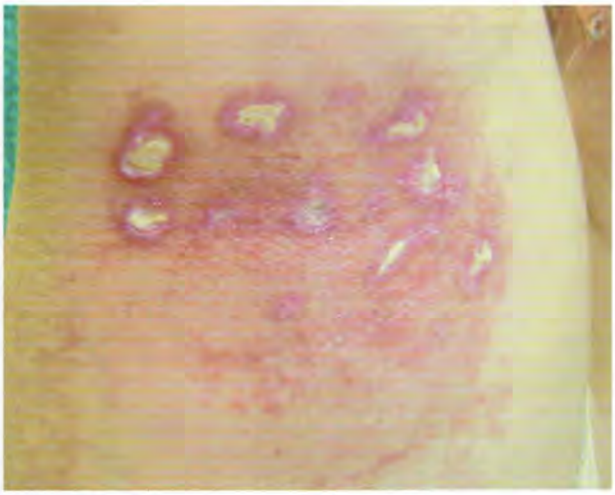


Fig. 3.11: Laser burns in axilla –Laser used for hair removal

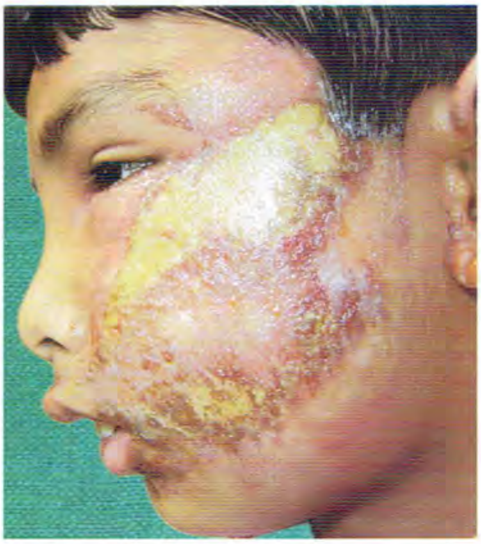


Fig. 3.12: Friction burns of face in a young boy

First Degree: Erythema without vesication

Second Degree: Vesication, loss of epithelium and inflammation

Third Degree: Destruction of papillary dermis

Fourth Degree: Destruction of whole dermis

Fifth Degree: Eschar formation of all superficial parts and muscle

Sixth Degree: Carbonization of whole limb

Dupuytren never described the morphology of third and fourth degree burns. After years of controversy, a general consensus dawned with the classification of Boyer and Ginnard in 1855, who categorized burns in three degrees based on erythema, vesication and eschar formation. Ironically it was almost identical to the postulation of Fabricus of 1610. This classification is still followed with slight modifications. Snonnenburg and Tschmarke in 1915 added fourth degree as carbonization which was added to the above classification. In 1944, Converse and Robb-Smith classified burns anatomically and described as Superficial (Partial Skin Loss), Deep (Total Skin Loss) and Mixed. This system was further refined by Jackson in 1953. He subdivided Partial Skin Loss into Superficial Skin Loss (Sup. SL) and Deep Skin Loss (Deep SL). He suggested that former heals from hair follicle remnants and latter heals from sweat gland and deep hair follicle remnants.

Burns in this context is basically involvement of epidermis and some amount of dermis and purely epidermal burn which is mostly seen in sun burn (Fig. 3.13) is not dealt in this context. The general consensus on classification of burn depth is on the basis of dermal thickness involvement which ranges from partial thickness to full thickness. Burns involving superficial papillary dermis is termed as superficial

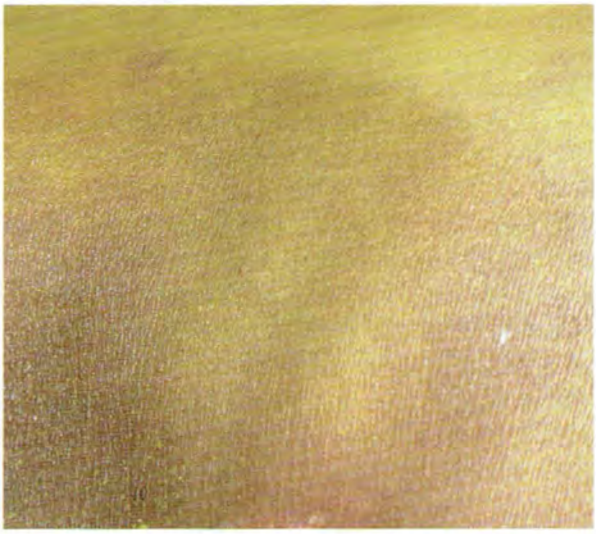


Fig. 3.13: Epidermal burn or sunburn

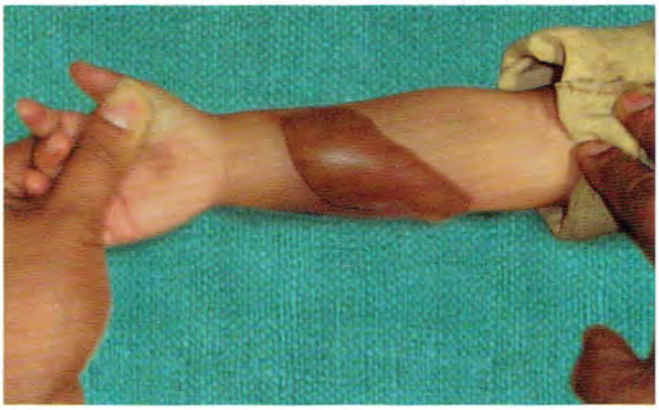


Fig. 3.14: Superficial partial thickness or superficial dermal burns showing blisters

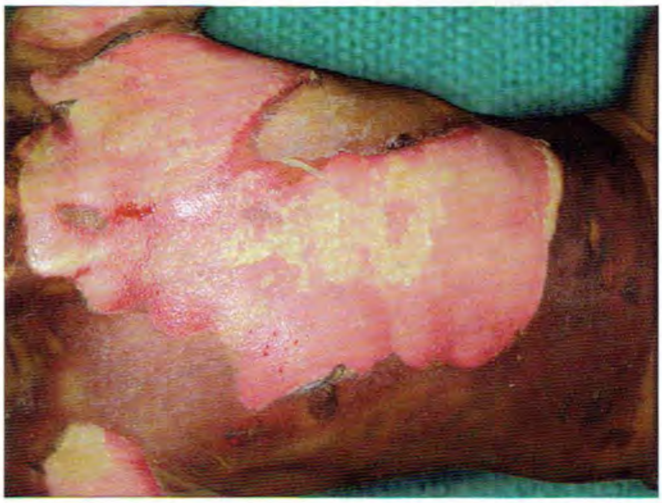


Fig. 3.15A: Superficial dermal burns over chest and abdomen after debridement of blisters with moist red look

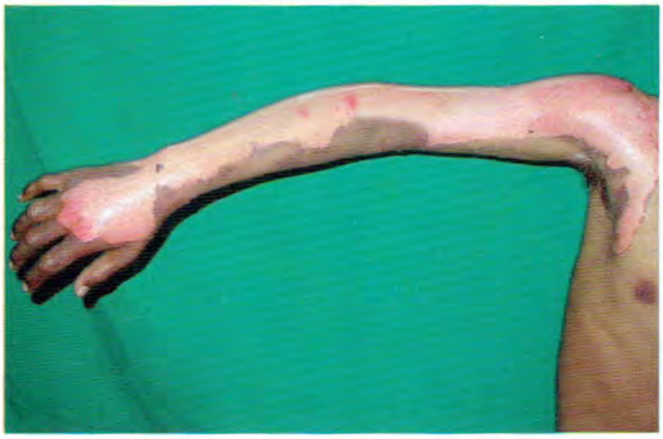


Fig. 3.15B: Superficial dermal burns involving arm after removal of loose dead skin. The areas blanch on pressure with brisk capillary refill

partial thickness burn and those extending into the deeper reticular dermis are called deep partial thickness burn. To categorize a burn as partial thickness some amount of dermis must be spared with its viable epidermal elements in hair follicle and glands, facilitating healing by epithelialization. On the contrary, a burn involving the whole of dermis is termed as full thickness burn which needs skin grafting as spontaneous epithelialization is not possible due to the lack of epidermal adnexal elements. A parallel terminology also used in this context is degree of burn. Epidermal burns are termed as first degree burn and as mentioned earlier are not considered in this context. Partial thickness burn is synonymous with second degree burn and is further sub-classified into second degree superficial dermal and second degree deep dermal based on the same criteria of involvement of dermal thickness mentioned above.

Full thickness burns are third degree burns. The modality of categorizing on the basis of depth is purely clinical and subjective based on the clinical picture of the wound and symptoms. In superficial partial thickness or second degree burns, vesication is primary feature (Figs 3.14 to 3.15B). This is due to subepidermal fluid collection and when deroofed, it exposes a glistening pink and wet dermis. Such bullae are not seen in deep partial thickness burn as the burnt epidermis and the reticular dermis sticks to the wound and give a waxy white appearance (Figs 3.16A and B). In full thickness burns, the whole devitalized skin is adherent as a parchment like eschar on the wound (Figs 3.17 and 3.18) with thrombosed vessels shining through the dead skin (Fig. 3.19). The depth of burn can also be judged by

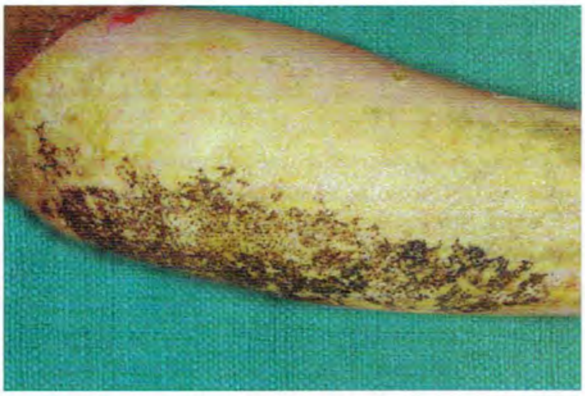


Fig. 3.16A: Deep dermal or deep partial thickness burns involving forearm with waxy white look

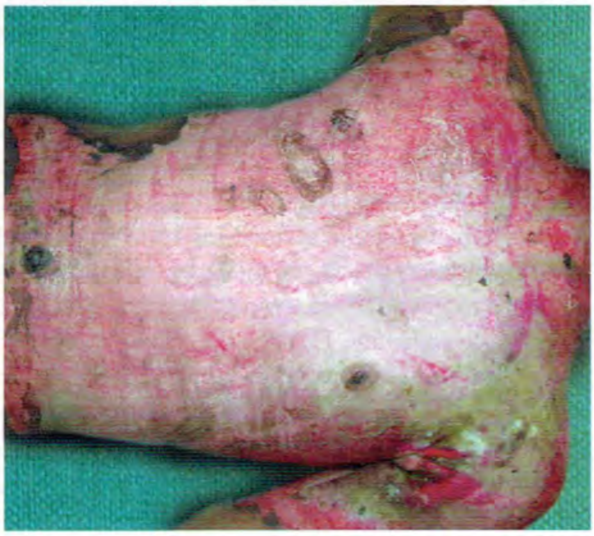


Fig. 3.16B: Deep dermal burns of chest and abdomen with mixed red and white wet look with areas that blanch on pressure but capillary refill is sluggish or absent

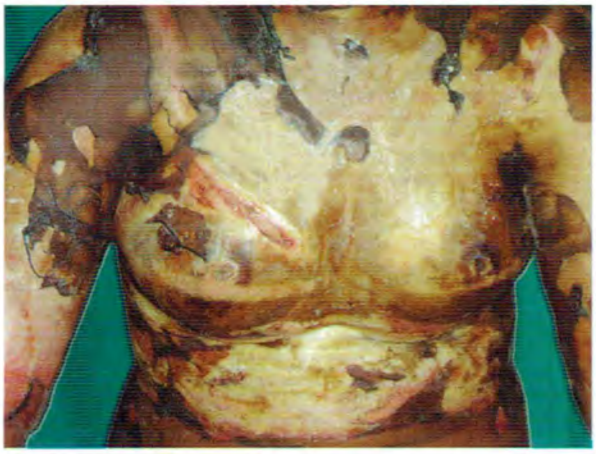


Fig. 3.17: Deep burns over chest and arms

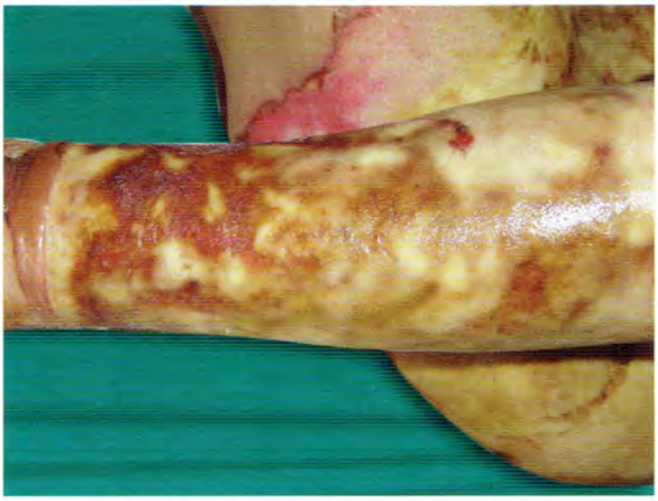


Fig. 3.18: Deep burns over arm and thighs

the involvement of hair follicles. In deeper burns there is easy pluckability of hair signifying their devitalization. Similarly, pain in the burn wound is inversely proportional to the amount of dermal involvement as nerve endings are destroyed in deeper burns. The pink appearance which blanches on pressure in a superficial burn indicates the rich capillary arborization in the superficial dermis and hence deep partial thickness burns are relatively pale or waxy white and do not blanch on pressure. The blanching test should always be done as a deeper burn can also be fallaciously erythematous due to fixation of erythrocytes leaked out of the destroyed capillaries. Such a picture is frequently seen in children who have thin skin. A subdermal burn damages muscle, bone, and interstitial tissue as is usually seen in electrical contact burns (Fig. 3.20). They are termed fourth degree burns.

A workable classification with interchangeable terms is given in Table 3.3. While using this system, it is prudent to note that burn wound is in a perpetual state of change depending upon tissue perfusion and infection. A wound categorized as superficial dermal on admission, may covert into deep dermal burn because of inadequate fluid resuscitation and wound infection. Hence, repeated and regular assessment is always rewarding diagnostically. The term 'eschar' needs to be defined, which is synonymous with third degree burn. But the waxy white layer seen in second degree deep dermal or deep partial thickness burn should also be regarded as "soft eschar". This layer is formed by dead papillary and reticular dermis and needs to be excised in tangential excision.

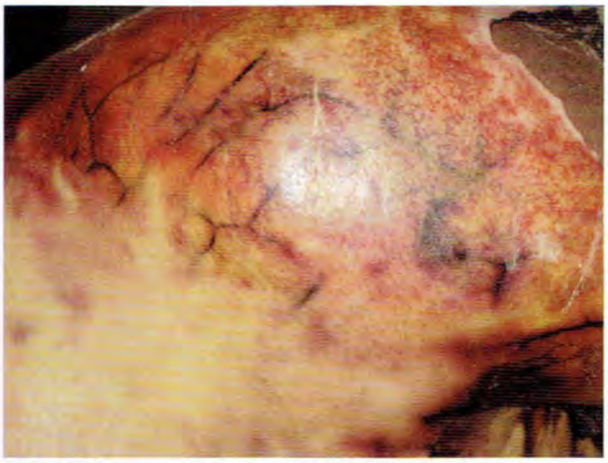


Fig. 3.19: Deep or full thickness burns over shoulder with dry translucent eschar with thrombosed vessels

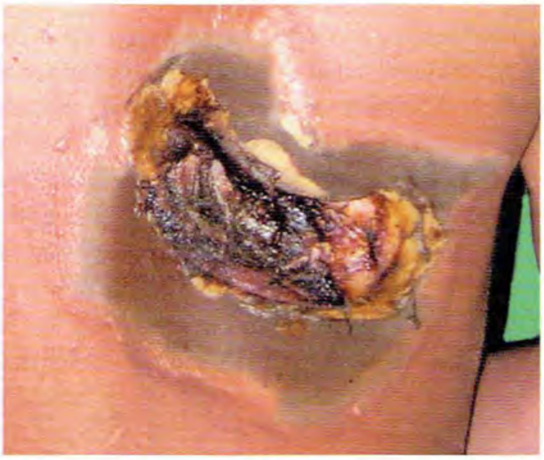


Fig. 3.20: Deep electrical contact burn involving skin, subcutaneous tissue, muscle and bone

It is obvious that this clinical classification system of burns, based on visual examination, is fraught with subjectivity and high experience requirement. There is an ongoing quest for an objective system to obviate these shortcomings. Numerous attempts using thermographic, infrared and ultraviolet imaging, radioisotope scanning, Doppler laser flux measurement and visual digital imaging have been reported.[8-13] Unfortunately, none have risen out of research journals into user friendly modality due to economic reasons.

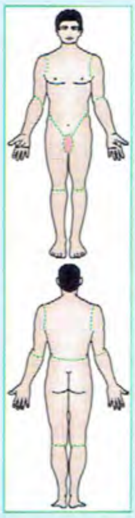
ESTIMATION OF BURN SIZE

The amount of body surface area involved in burns, expressed as a percentage of the total body surface area (%TBSA), is the key to the estimation of fluid requirement to treat shock and is also an important prognostic factor. The extent of burn is estimated by Wallace's Rule of Nine. According to this system head and neck account for 9%, each upper limb and each lower limb constitute 9% and 18% respectively. The anterior and posterior trunk constitutes 18% each and external genitalia account for 1%. The estimation is different in children owing to the larger proportion of the head. Hence in a child, head and neck account for 18% and the lower limb constitute 13.5% each. Proportion of upper limb and trunk remains same as adult. A more accurate estimation system is the Lund and Browder Chart which can be used for all age groups and also compensates for variable body size and shape (Table 3.4). Estimation of extent of scattered and irregular areas in all age groups is done by Rule of Palm or Hand. The palm of an individual with fingers and thumb adducted account for 1%.

Table 3.3: Clinical classification of burns

Burns	*Involvement*	*Features*	*Depth*
First degree (Sun Burn)	Epidermis	Erythema only. Heals in < 5 days	Partial thickness
Second degree - *Superficial dermal or Superficial partial thickness*	Epidermis and papillary dermis	Extremely painful Blister formation. Wet, glistening pink Heals in 10-15 days.	Partial thickness
- *Deep dermal or deep partial thickness*	Epidermis, papillary dermis and part of reticular dermis	Less painful, waxy white appearance. Heals in 3-5 weeks.	Partial thickness
Third degree	Entire thickness of skin	Painless Inelastic, translucent, parchmentlike eschar with visible thrombosed veins Require skin grafting.	Full thickness
Fourth degree	Skin, subcutaneous tissue, muscle, carbonization of limb or gangrene	Thick eschar to gangrene	Full thickness

Table 3.4: Lund and Browder's chart for estimating area of burns

Area	*Birth to1 year*	*1 to 4 years*	*5 to 9 years*	*10 to 14 years*	*15 years*	*Adult*	*2nd°*	*3rd°*	*TBSA*
Head	19	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Anterior trunk	13	13	13	13	13	13			
Posterior trunk	13	13	13	13	13	13			
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Genitalia	1	1	1	1	1	1			
Right upper arm	4	4	4	4	4	4			
Left upper arm	4	4	4	4	4	4			
Right lower arm	3	3	3	3	3	3			
Left lower arm	3	3	3	3	3	3			
Right hand	2.5	2.5	2.5	2.5	2.5	2.5			
Left hand	2.5	2.5	2.5	2.5	2.5	2.5			
Right thigh	5.5	6.5	8	8.5	9	9.5			
Left thigh	5.5	6.5	8	8.5	9	9.5			
Right leg	5	5	5.5	6	6.5	7			
Left leg	5	5	5.5	6	6.5	7			
Right foot	3.5	3.5	3.5	3.5	3.5	3.5			
Left foot	3.5	3.5	3.5	3.5	3.5	3.5			
Total:	100	100	100	100	100	100			

CLASSIFICATION ACCORDING TO SEVERITY OF BURNS (TABLE 3.5)

The severity categorization of burn is done by taking into consideration extent and depth. This classification is useful for establishing admission criteria and is indispensable in triaging in event of a fire disaster. The categorization is as follows:

Critical Burn

a. Partial thickness burn >25%TBSA in adult, >15%TBSA in children and > 5%TBSA in infants.
b. Full thickness burn >10%TBSA in adults and > 3%TBSA in children and infants.
c. Burns of hand, face and perineum
d. Burns with associated head injury, thoracic or abdominal trauma, old age and preexisting systemic illness.
e. Burns with associated inhalation injury
f. Electrical burn.

Table 3.5: Classification of burns as per their severity

Grade	*Criteria*
Critical burns	Partial thickness burns > 25% TBSA (total body surface area) Full thickness burns >10% Burns of special sites like hands, face and perineum. Burns with associated injury or any pre-existing illness and old age. Electrical burns. Burns complicated by respiratory tract involvement.
Moderate burns	Partial thickness burns 15-25% TBSA Full thickness burns 1-3% TBSA
Minor burns	Partial thickness burns <10-15% TBSA.

Moderate Burns

a. Partial thickness burn 15-25%TBSA in adults and 10-15%TBSA in children and infants.
b. Full thickness burn 3-10%TBSA in adults and 1-3%TBSA in children and infants.

Minor Burns

a. Partial thickness burn< 15%TBSA burn in adults and < 10%TBSA in children and infants.
b. Full thickness burn <3%TBSA in adults and up to 1%TBSA in children and infants.

Grading of Burns

A further refinement to this system was propounded by Kumar (1993) by including the percentage of deep burn as an additional criterion (Table 3.6).[14] He included deep partial thickness and full thickness burn as "Deep" burn and estimated Burn Depth Index (DI) by calculating the fraction of "Deep" burn out of the total extent of burn.

$$\text{DI} = \frac{\text{Total \% of Deep Burn}}{\text{Total \% TBSA Burn}}$$

A severity and prognostic grading taking into consideration the extent, depth and DI is given in Table 3.6. Unlike the previous grading this system helps determine the prognosis, quantum of deformity and the need for skin grafting.

Although the basic purpose of these classifications is to guide the treatment modality, but most often burns are still categorized simply as superficial and deep where early excisional surgery is not routinely practiced.

A knowledge of the etiology and mode of burn injury is important for devising preventive strategies whereas classification based on extent and depth guides management protocol and determines prognosis.

Table 3.6: Prognostic grading of burns as per their severity

Grade	*Criteria*
I Minor Burn	a. Erythema <15% TBSA (<0.5 DI) <15% TBSA (>0.5 DI)
II Moderate Burn	a. 15-30% TBSA (<0.5 DI) b. 15-30% TBSA (>0.5 DI)
III Major Burn	a. 30-65% TBSA (<0.5 DI) b. 30-65% TBSA (>0.5 DI) c. >65% TBSA (<0.5 DI) d. >65% TBSA (>0.5 DI) e. Burns of hands, face and perineum. Electrical burns. Associated injury Head injury, fracture, Chest and abdominal injury Pre-existing illness and old age Inhalation Injury

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Chapter

4

Pathophysiology of Burn Shock

Arvind Vartak

INTRODUCTION

Burn injury is the commonest form of injury that afflicts the human beings. Extensive burns are the severest form of trauma. It is the most painful of all injuries, most complicated to treat, most expensive to manage and leaves behind permanent memories, both physical and psychological. Over the last several decades, better understanding of physiological changes which occur following a burn injury has led to great improvements in treatment. This has significantly improved the results by reducing the morbidity and mortality in burn injured patients.

Following a severe burn, the patient passes into a state of shock.[1] The success in adequate management of shock can be achieved with anticipation, recognition and suitable treatment of shock. Hypovolemic shock is a disparity between the circulating blood volume and the capacity of vascular tree (the intravascular compartment). Neurogenic shock appears as a state of profound mental and physical depression consequent to severe physical injury and/or an emotional disturbance (Stedman's 1976).[2] In case of burn injury, this may be a result of intense pain associated with emotional disturbance immediately after the burn injury occurs. It is characterized by pallor, dizziness, restlessness and rarely unconsciousness. This state passes off spontaneously and usually is not noticed by the time the patient reaches a burn care facility. It does not need any specific treatment.

PATHOPHYSIOLOGICAL CHANGES FOLLOWING BURNS

Hypovolemic shock appears as a result of loss of liquid component of the blood (plasma) in to the tissues leading to edema formation in surrounding tissues. After burn injury there is an immediate transient decrease in the blood flow in the involved tissues. This is followed by pronounced arterial vasodilatation in patent microcirculation. Cotran (1965)[3] demonstrated through the electron micrographic studies that the burn induces an endothelial cell gap in capillary microvasculature. This leads to increased capillary permeability from intravascular to extravascular compartment. Arturson (1966)[4] demonstrated the extravasations of fluorescein-labelled dextran with molecular weight around 150,000 from post capillary venules within 10 minutes after a thermal injury. This leads to burn edema. In extensive burns, due to large volume of fluid lost from intravascular compartment, systemic hypovolemia develops. This causes fall in the pressure at the arterial end of capillary network. In an untreated or inadequately treated burn injury edema formation is retarded. After adequate resuscitation is carried out, the circulation is restored. With restoration of the blood flow, edema formation begins as a result of gradual, prolonged fluid flux into interstitial spaces.

The changes in a burn wound take place due to the effect of the heat and superimposed by acute inflammatory reaction (Arturson 1996).[5] There is rapid edema formation which reaches its peak in 1 to 3 hours post burn. Increased permeability may prolong up to 16 hours post burns. Severe interstitial edema formation may cause significant ischemia due to the compression of subdermal vessels and can lead to progression of necrosis. This leakage from intravascular to extravascular compartment may continue for a long time, even for 24 hours. Baxter (1967, 1974) [6,7] feels that the increased capillary permeability following a burn injury continues for about 24 to 36 hours and recommends that the colloids should be avoided for first 24 hours.

Capillary permeability, following direct exposure to heat and in response to certain substances released in the tissues which include histamine and serotonin, increases severely, leading to loss of fluid from intravascular compartment to extravascular compartment. These changes can occur even in unburnt tissues. This increased permeability in unburnt tissues like skin and muscles may remain elevated for as long as 12 hours after the burn injury for the molecules of the size of albumin. The sustained increase in water content and elevated lymphatics flow in these tissues is probably caused by sustained hypoproteinemia (Arturson 1996).[5]

In superficial dermal burns, the fluid eventually exudes out of the wound in the form of blisters. There is preponderance of albumin in this exudate due to smaller size of albumin molecules. This permits their escape into extravascular compartment more easily. Sometimes clotting occurs in blister fluid indicating that large fibrinogen molecules have also leaked out of the vascular tree. This loss of protein-rich fluid from plasma after burn leads to a clinical condition of shock.

The protein content of the exudate may be as high as 80% of plasma levels (Muir, Barkley and Settle (1987).[8]

	Plasma	*Blister Fluid*
Albumin	3.9 gm%	3.7 gm%
Globulin	2.6 gm%	1.3 gm%
Total Proteins	6.5 gm%	5.0 gm%

After 24 to 36 hours, the capillary endothelial integrity returns spontaneously. The large molecules of colloids which leaked into the interstitial spaces can not be brought back into the circulation. This additional colloid load has to be handled by the lymphatics.

Under normal circumstances, the lymph returns to the circulations in volume comparable to intravascular pool. About 50% of circulating plasma protein is carried daily by lymphatics. Following a burn injury, quite obviously, the lymphatic system is put to a very severe increase in its work load and can not function efficiently. This occurs due to increase in the volume as well as the process of coagulation due to leakage of fibrinogen and its products in interstitial spaces leading to lymphatic obstruction (Arturson 1996).[5]

Extensive extracellular edema after the burn injury may occur as a result of increased capillary hydrostatic pressure. Reduction in protein concentration of plasma causes reduction of colloidal osmotic pressure and together with lymphatic blockage and increased permeability of capillaries leads to leakage of protein rich fluid into the extravascular compartment. Dilatation of capillaries causes a dramatic fall in peripheral resistance and increase in the blood flow. The altered endothelial permeability leads to the leakage of larger protein molecules into the interstitial compartment (Arturson 1996).[5]

In the absence of treatment, the natural defense mechanisms of the body counter the development of shock in the following ways:

1. Proportionately less loss of protein than that of electrolytes. This leads to relative increase of plasma osmotic pressure, which reduces further leakage.
2. By withdrawing fluid from extracellular space of undamaged tissues which may be result of increased osmotic pressure.
3. By general visceral vasoconstriction reducing the volume of vascular compartment.
4. Development of intense thirst leads to increased ingestion of fluids and rapid absorption from the gut.

By these mechanisms, the body may be able to compensate for the rate of fluid loss in minor and moderate burns. The patient may pass safely through the shock phase. However, in extensive burns, the rate of loss outweighs these in-built defense mechanisms and the plasma volume continues to fall. When untreated or inadequately treated, the patient may progress into a state of irreversible shock.

Edema formation is retarded in larger injury as a result of systemic hypovolemia until adequate fluid resuscitation is carried out. On many occasions, loss of plasma volume occurs in excess of replaced fluid in spite of massive infusion over several hours.

In minor and moderate burns, it may be possible to achieve the replacement of lost fluid by oral ingestion alone. In extensive burn injury, however, the rate of loss of fluid from the intravascular compartment is so high that it may not be possible to replace the fluid at the rate at which it is lost by oral route alone. Replacement by intravenous route is then necessary. Adults with 25% or more Burn Surface Area (BSA) require intravenous replacement of lost fluids. Children and elderly patients are likely to require aggressive resuscitation even with a burn injury as little as 10% BSA.

It will be useful to understand the fluid shift in normal tissues. Under physiological conditions, the high pressure in the arterial end of capillaries causes the filtration of plasma into the tissue interstitial spaces. At the venous end of capillaries this fluid is partially reabsorbed and returns to the circulation. Remaining fluid is handled by the lymphatic system which drains finally to the venous system.

Burn injury is only to the skin; however secondary involvement of all organs can occur as a result of reduced tissue perfusion due to a state of shock. In untreated severely burned patients the changes become more severe and widespread and finally lead to death due to multiorgan failure.

EFFECTS OF OLIGEMIA ON VARIOUS ORGANS (TABLE 4.1)

As a result of reduction of plasma volume, vasoconstriction and decrease in venous return, there is a marked **fall in cardiac output** in patients with severe burn. There is severe tachycardia. With proper fluid replacement, the cardiac output promptly returns to normal. Without adequate fluid therapy the cardiac output continues to fall at a slower rate until death occurs as a result of multi-organ failure.

As a result of vasoconstriction and increased heart rate, **blood pressure** is maintained till late and **is not a reliable guide** for the assessment of state of shock. Moreover, in these patients, due to formation of severe edema, blood pressure readings cannot be taken accurately.

Table 4.1: Pathophysiologic effects of major burn shock

System	*Early*	*Late*
Cardiovascular	↓ Cardiac Output (CO) due to decreased circulating blood volume, impaired cardiac contractility ↑ Systemic vascular resistance (SVR)	↑ CO due to sepsis ↑ CO may be 2-3 times > baseline hypertension, tachycardia ↑ SVR
Respiratory	Upper airway obstruction due to edema Lower airway obstruction due to edema, bronchospasm, particulate matter ↓ FRC ↓ Pulmonary compliance ↓ Pulmonary edema (as a consequence of overzealous resuscitation)	• Bronchopneumonia • Tracheal stenosis, laryngeal damage (as complication of intubation) ↓ Chest wall compliance
Renal	↓ Renal blood flow ↓ GFR: due to a. ↓ Circulating blood volume b. Myoglobinuria c. Hemoglobinuria	↑ Renal blood flow ↑ GFR 2° to ↑CO Tubular dysfunction
Hepatic	↓ Function due to ↓ circulating blood volume, hypoxia, hepatotoxins	↑ Functions due to hyper metabolism, enzyme induction, ↑ CO ↑ Functions due to sepsis-drug interaction. Hepatitis
Coagulation and Hematology	Hemoconcentration Hemolysis ↓ Platelets ↑ Fibrin split products, consumptive coagulopathy, anemia	Anemia
Infection and immunity	Impaired immune function (burn wound sepsis, pneumonia)	Multiple organ failure, endotoxemia
Neurologic	Encephalopathy Seizures ↑ ICP	Encephalopathy Seizures ICU psychosis
Metabolic	↓ ionized calcium Impaired thermoregulation	↑ metabolic rate ↑ CO_2 product and O_2 utilization.
Gastrointestinal	Stress ulceration (Curling's ulcer) Impaired intestinal barrier function Endotoxemia	Stress ulceration (Curling's ulcer) Impaired intestinal barrier formula Endotoxemia
Skin	↑ Heat, fluid, electrolyte loss	Contractures, scar formation
Pharmacokinetics	Altered volume of distribution	Tolerance to narcotics, sedatives, Enzyme induction, altered receptors. Drug interaction.

↓, decrease in; ↑, increase in; CO, Cardiac output; FRC, Functional residual capacity; GFR, glomerular filtration rate; ICP, intracranial pressure; ICU, intensive care unit

Adopted and modified from: McLennan N, Heimbach DM, Cullen BF. Anesthesia for major thermal injury. Anaesthesiology 1998, 84: 749-770.

In the **intestines**, vasoconstriction may be so intense that the blood flow may be inadequate to satisfy the needs of cells of capillary walls. Absorption of fluid from intestine cannot occur, peristalsis ceases and **paralytic ileus** develops. At this stage, the patient who is intensely thirsty starts vomiting after any attempt at drinking fluids and this important defense mechanism fails. Presence of peristalsis indicates that the visceral circulation is adequate. Similarly in the stomach, the intense vasoconstriction and hemoconcentration resulting in micro-thrombi may lead to acute ischemic damage to gastric and duodenal mucosa resulting in acute mucosal lesions called 'Curling's ulcer".

Brain continues to receive significant contribution of blood flow for a considerable time and consciousness **is not lost** till late.

There is severe reduction in microcirculation in **lungs** though rapid and continuous blood flow in larger vessels is noticed. The ventilation rate increases and is usually proportional to the severity of burn injury and declines gradually. Oxygen consumption also increases but is not affected by the ventilation rate if the respiratory exchange is not impaired. However, in case of inhalational injury the oxygen tension decreases and the blood gas changes are noticed. This is discussed in detail in chapter on Respiratory tract injury in burns. **Interstitial edema** is present in varying degrees which later leads to a classical condition called **ARDS** (Acute Respiratory Distress Syndrome). Both these conditions lead to inadequate oxygen transfer resulting in marked hypoxia in various organs.

Of all the viscera, the **kidneys** are most likely to be damaged by persistent shock. Secretion of urine is a valuable guide to renal function and indirectly to the blood flow of kidneys and thus to the tissue perfusion is general. During the shock phase, there is a marked rise in Antidiuretic Hormone (ADH). This results in conservation of body fluids when oligemia is present. Antidiuresis may be the combined effect of ADH, hypovolemia and reduction of plasma flow due to release of vasoactive substances (Wilkinson, 1966).[9] After a prolonged, uncontrolled shock, renal failure ensues. After about 48-72 hours, if hypovolemia is adequately corrected, reabsorption of interstitial fluids starts, the level of ADH falls and the phase of diuresis starts.

Liver, pancreas, endocrines and in fact **all other organs** are directly or indirectly affected due to the effects of oligemia, fall in cardiac output and vasoconstriction. The liver enzymes (SGOT, SGPT and Alkaline phosphatase) rise promptly following burns, peak at 2-3 days and persist at high levels till several days post burns due to the liver damage caused by oligemia.

In the **endocrine** system there are variable changes in the different hormonal levels. There is decrease in insulin production in early post burn period lasting for 2 to 3 weeks. Glucagon is elevated but fasting blood sugar is not grossly increased. Growth hormone increases in the first 2 weeks and then decreases to a lower level which is still above normal for another 6 to 8 weeks. This can lead to slight increase in blood glucose levels. ACTH level increases proportional to burn size, especially in children. Cortisol levels increase two to four fold. The Gonadotrophic hormones are only slightly affected.

The effects of thermal injury on the microvasculature of the skin are discussed in pathophysiology of the Burn Wound.

Anemia in Burn Patient

The term anemia generally pertains to reduction below normal of the concentration of oxygen transporting capacity of a designated volume of blood and is generally expressed as the amount of hemoglobin in 100 ml of blood (Stedman 1976).[2]

Following extensive burn, plasma is rapidly lost from intravascular compartment to extravascular compartment and causes significant rise in hemoglobin and hematocrit levels. This slows down the blood circulation particularly in the zone of stasis leading to local tissue anoxia (Jackson 1953).[10] Hence, blood transfusions are not advisable at this stage. Hemoconcentration can have deleterious effect on the zone of stasis where the circulation in the tissues has already slowed down.

After initial rise of hemoglobin and hematocrit levels, a patient with extensive burn injury becomes rapidly anemic. Lowered hemoglobin can lead to tissue hypoxia, retard wound healing and hamper the 'take' of skin grafts.

In a burn patient, anemia can develop due to various causes/mechanisms (Davies 1982).[11]

1. Red cells destruction as a result of direct heat during the process of burning.
2. The red cells which are not completely destroyed but are exposed to heat are rendered unduly fragile. They are sooner or later removed from the circulation earlier than the expected balance life span of these RBCs.
3. Overzealous resuscitation leads to over-hydration and consequent hemodilution gives rise to a false anemic state.

4. Bacterial toxins can cause hemolysis, particularly the invasive infection by various hemolytic organisms sometimes leading to massive red cells destruction.
5. Severe hemorrhage at the dressing changes (particularly when the dry dressing is used) during removal of adherent dressing at the stage when the granulation tissue in the wound has started developing after the necrotic tissue has come out.
6. Thrombocytopenia leading to increase in the bleeding tendency all over the body which may result in bleeding from the wound, mucous membrane, lungs, gastrointestinal tract and in fact anywhere in the body.
7. Inadequate assessment of blood loss and resultant inadequate blood transfusions during operative procedure like escharectomy, debridement of granulation tissue, skin grafting operation, primary or secondary excision of the burn wound, etc.
8. Poor nutritional status of patients with extensive burns.
9. Biochemical and morphological alterations in red cells and hemoglobin lead to their reduced efficiency in oxygen transport.

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Chapter
5

Pathophysiology of Burn Wound

Arvind Vartak

INTRODUCTION

The skin is the largest organ in the body. It is made of epidermis and the dermis. In the dermis there is ingrowth of epidermal elements like sweat glands, sebaceous glands and hair follicles (Fig. 5.1).

The epidermis consists of stratified squamous epithelium. The deepest layer consists of the tall columnar cells. As they approach the surface they flatten. The outer layer is continuously shed off as dead cells and forms a keratin layer which is continuously replaced from underneath.

Dermis is formed mainly by the fibrous connective tissue and ingrowths of various epidermal structures from out side, namely, sweat glands, sebaceous glands and hair follicles. Blood flow to the skin comes through capillaries arising from subdermal plexus of blood vessels which bring nutrients to the skin. It also contains nerve endings which provide sensation to the skin.

The characteristics of the skin vary from place to place depending upon the function of the particular part of the body. Thus, the skin of upper eyelid is very thin while that of the sole is very thick. It can be different even in the same part of the body, e.g. the skin of the palm is thick while that on the dorsal aspect of hand is thin and very loose so that free movements of the fingers and the hand are possible.

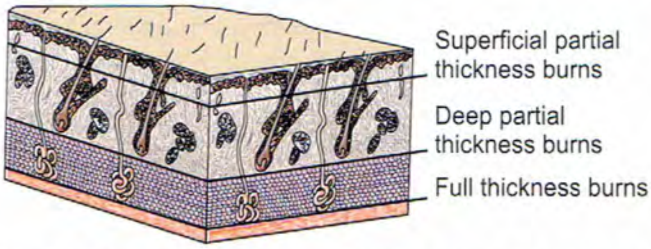

Superficial partial thickness burns
Deep partial thickness burns
Full thickness burns

Fig. 5.1: Diagrammatic representation of anatomy of the skin and classification of burn injury in relation to the depth of burn-superficial partial thickness, deep partial thickness and full thickness

Heat destroys a greater or lesser depth of the skin, depending upon the intensity and the duration of exposure. When the burn injury causes partial thickness damage, the epithelium from the epidermal appendages grows to the surface of viable dermis underneath the devitalized layers. The dead tissue separates and is shed off after the healing underneath is complete.

Greater the depths of the burn, lesser are the epidermal structures available to grow and develop a new epidermis. Thus, the superficial burns heal rapidly while the deeper partial thickness burns take a longer time to heal; may be even 3-6 weeks. Full thickness burns, however, heal either by contraction, if very small or with a contracture formation if large and opposite joints. They may not heal at all over rigid areas of bone, e.g. skull unless surgical intervention is done in the form of skin grafting.

The burn wound, although it damages only the skin cover of the body, is the cause for a variety of widespread complications in the body involving almost all organs systems, initially as a result of oligemia and later due to septic complications.

FUNCTIONS OF THE SKIN

The skin acts as a protective barrier and prevents invasive infection. When this barrier in broken, the microorganisms from outside can invade into the body. When large areas of skin are damaged, they can lead to bacteremia or even septicemia.

Skin also helps in retaining body fluids. When large areas of skin are damaged, plasma leaks out. This can result in a state of hypovolemic shock. This loss can sometimes be so large that a correction by intravenous infusion becomes necessary and the replacement of fluids by oral route alone is not enough. This lost fluid contains various salts leading to electrolyte imbalance.

Body temperature is regulated by dilatation or constriction of blood vessels in the dermis. Vasoconstriction reduces the heat loss while vasodilatation increases it. Sweating helps in cooling the body in a hot climate.

The sebaceous glands secrete a fatty substance which lubricates the skin, keeps it soft and protected from water.

The nerve endings are the defence mechanism of the body through various sensations, which perceive touch, pain, pressure and changes in temperature, etc.

Skin also helps in immune responses of the body. Rickatts et al (1951)[1] also postulated that the skin has self-sterilizing ability.

The transfer of thermal energy to the skin occurs in various ways like conduction-in a case of contact burn, convection-when there are currents of hot air or liquid and radiation-by transfer of heat without involving the medium through which it occurs, like in case of sunburn or from bonfire (Williams and Philips 1996).[2]

Mortiz and Henriquez (1947) [3]demonstrated that the transfer of heat from the surface to the deeper layers leads to cellular necrosis depending upon the temperature and the duration of exposure.

The Heat Required to Cause Skin Burns

Full thickness
At 44°C in 6 hours.
At 52°C in 6 minutes.
At 59°C in 1 minute.
At 62°C in 30 seconds.
At 67°C in 20 seconds.
At 71°C in 10 seconds
Partial thickness
At 70°C in 1 second.

Ofeigsson (1965) [4]conclusively demonstrated that use of water as a cooling agent for the burn wound is beneficial. His animal experiments showed that the general condition of the treated animals was far better than that of controls. There are much less secondary infections. Water cooling arrests and causes retrogation of the degenerative process of the skin and underlying tissues, thus significantly shortening the period of healing. He also showed that the superficial temperature of the scalded skin of controls takes several minutes to fall and thus causing longer exposure to heat (longer than what we believe.) He also warned that extensive burns treated with ice water can cause hypothermia. This is likely to be true in cold countries and in Northern India in winter.

Cotran (1965)[5] carried out electron micrographic studies and showed that a burn induces endothelial cell gaps in the capillary microvasculature. This leads to increased capillary permeability from intravascular compartment to extravascular compartment leading to burn edema.

When injured, the skin repairs itself by:

1. The growth of the epidermis from the margins of unburnt skin.
2. Epidermal growth from intradermal epidermal adnexal elements, namely, sweats glands, sebaceous glands and hair follicles.

HISTOPATHOLOGY OF BURN WOUNDS

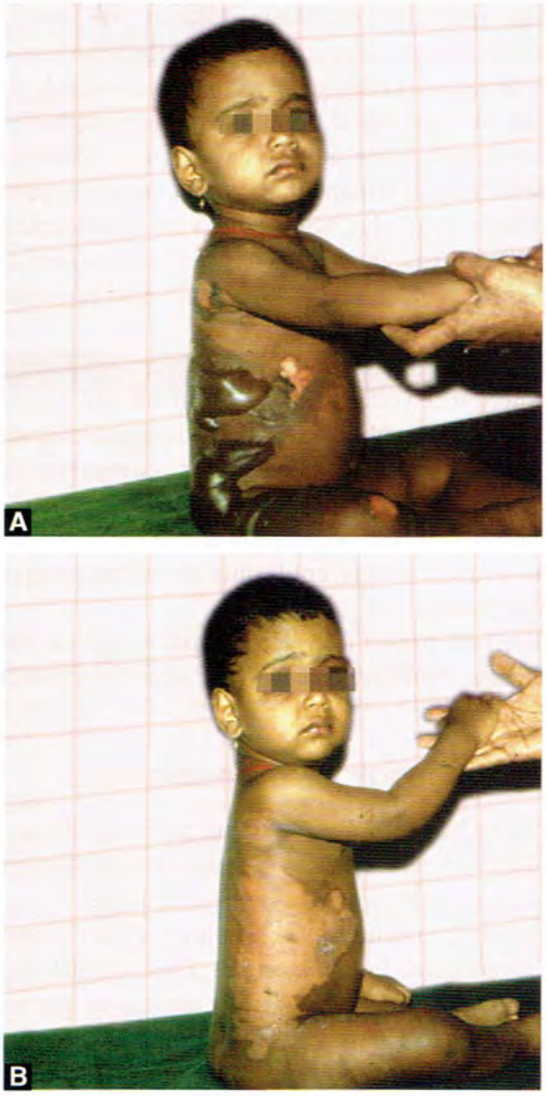
Burn injury has been classified into partial thickness burn and full thickness burn in relation to the anatomical involvement of the layer of skin involved (Fig. 5.1).

In a partial thickness burn, whole of epidermis and part of dermis are burnt (Figs 5.2 and 5.3). The epithelial elements from the remaining part of dermis grow over the surface between the viable dermis and the superficial necrotic layer which finally leads to healing. The necrotic layer separates and is finally shed off.

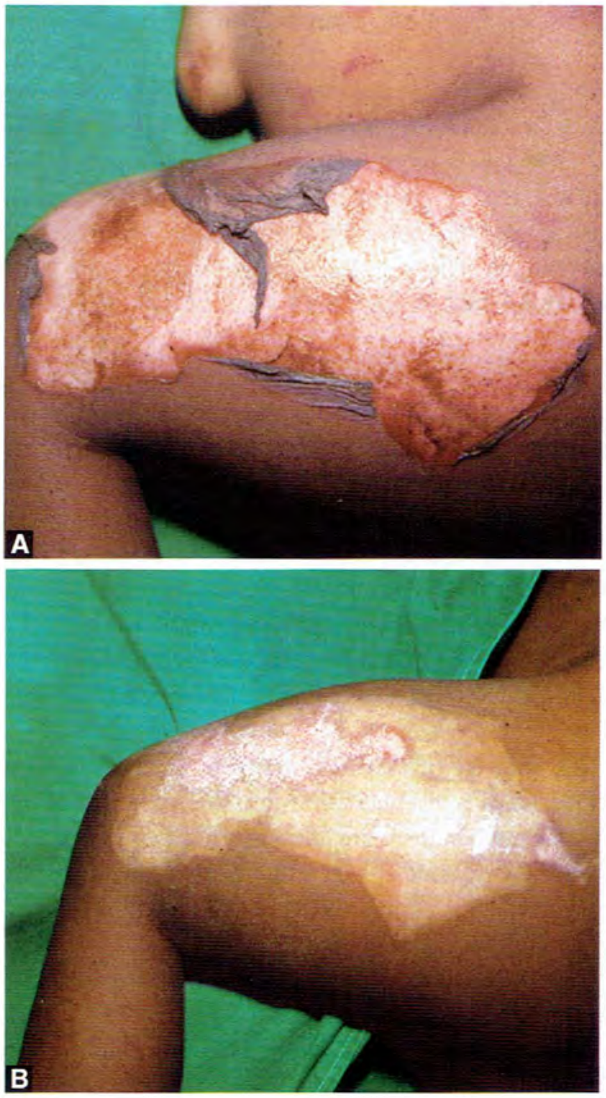
The partial thickness burn is further classified as superficial partial and deep partial thickness burns. Superficial partial thickness burns heal rapidly in up to 3 weeks due to greater availability of epidermal adnexal elements in them.

In deep partial thickness burns, however, these epidermal elements are much less in number and obviously take a longer time to heal, may be up to 6 weeks (Figs 5.4 and 5.5).

In a full thickness burn, entire thickness of the skin involving whole of epidermis and dermis is damaged. The epithelial elements are completely destroyed. Healing cannot take place at all. However, in small full thickness burn wounds, healing can occur from peripheral unburnt skin and wound contraction. Larger burns can heal only after skin grafting (Fig. 5.6).



Figs 5.2A and B: Superficial burns. (A) Note the characteristic large blisters. (B) Rapid healing in 10 days post burn



Figs 5.4A and B: Deep burns. (A) Appearance 2 days post burn. (B) Complete healing in three weeks

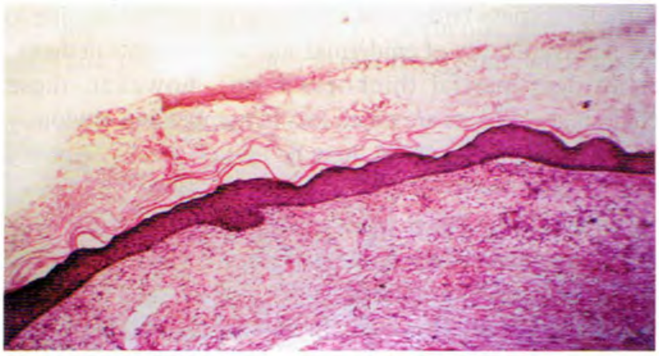


Fig. 5.3: Histological appearance (20X) of burn wound treated with BPP (Boiled Potato Peel) bandage. 11th post burn day. Re-epidermalization is quite evident between the slough on the surface and viable dermis underneath

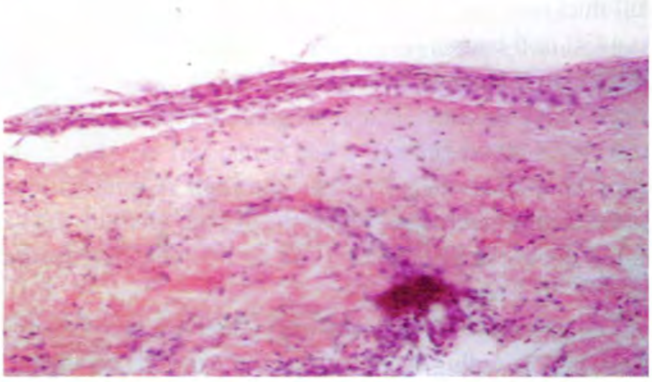


Fig. 5.5: Histological appearance (40X) of burnt skin on 2nd post burn day shows thin sloughed off epidermis. There is marked dermal edema with neutrophilic infiltration all over

Following a full thickness burn, the dead and necrotic skin gradually gets separated from underlying viable tissues and is shed off. The resulting raw area gets covered with granulation tissue, a tissue developed from the capillary network (Fig. 5.7), over which a skin graft can be applied for complete healing of wound. Immediately underneath the granulation tissue a thin fibrous layer is formed which gradually undergoes thickening.

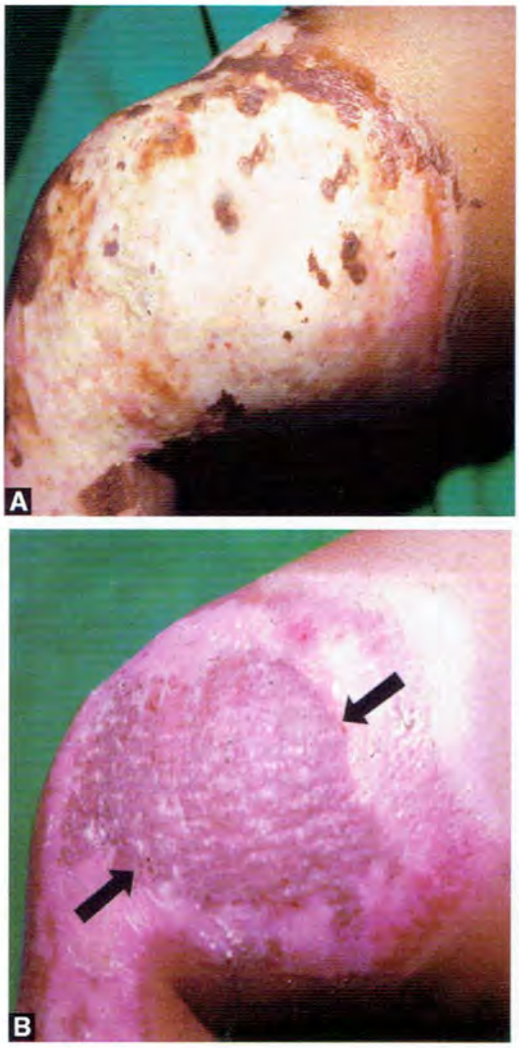
Figure 5.7 illustrates the character of granulation tissues under microscope with resultant micro pockets (shown with an arrow). These need to be cleared by debridement of the granulation before the application of the skin graft to ensure better 'take' of the graft (Fig. 5.8).

Jackson (1953) [6]described 3 zones in the damaged burn tissue (Fig. 5.9).

1. Zone of coagulation is the central zone with complete coagulative necrosis of the tissue cells which become completely nonviable and obviously cannot recover with any treatment.
2. Zone of stasis is on the periphery of zone of coagulation where a significant reduction in microcirculation occurs following damage to the tissues and its arterioles, venules and capillaries. The circulation is sluggish. This zone can recover after adequate resuscitation and proper wound care. This can result in healing of the burn wound. With inadequate resuscitation initially and due to invasive sepsis later, this zone can get converted into a zone of coagulative necrosis.
3. Zone of hyperemia is the outermost zone, peripheral to the zone of stasis, due to intense vasodilatation. This eventually recovers following adequate resuscitation, with passage of time.

Zawaki (1974) [7]demonstrated that the zone of stasis begins to recover in about 16 to 24 hours post burn. Oxygen consumption returns to this zone as early as 4th post burn day and exceeds normal by 7 to 14 days when resuscitation is adequate and drying of the wound is prevented. When a burn wound is exposed to air, dehydration of tissues occurs and the process of reversal of stasis is minimum or absent. Deep partial skin loss or whole skin loss occurs when the dermis is allowed to dry. The retention of blister skin or coverage with allograft or xenograft prevents dehydration of the dermis and helps the healing process favorably (Fig. 5.10).

Zawaki (1987)[8] showed that when the drying of a burn wound is prevented, epithelium migrates towards the surface around the necrotic hair follicles (Fig. 5.2) soon after the circulation in the viable tissue returns. He also showed that



Figs 5.6A and B: Full thickness burns. (A) Appearance 1 week post Burn. (B) Healing after skin grafting; skin graft marked by arrows

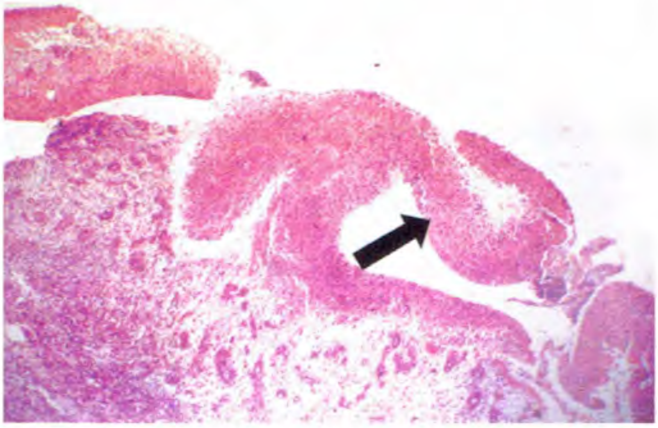
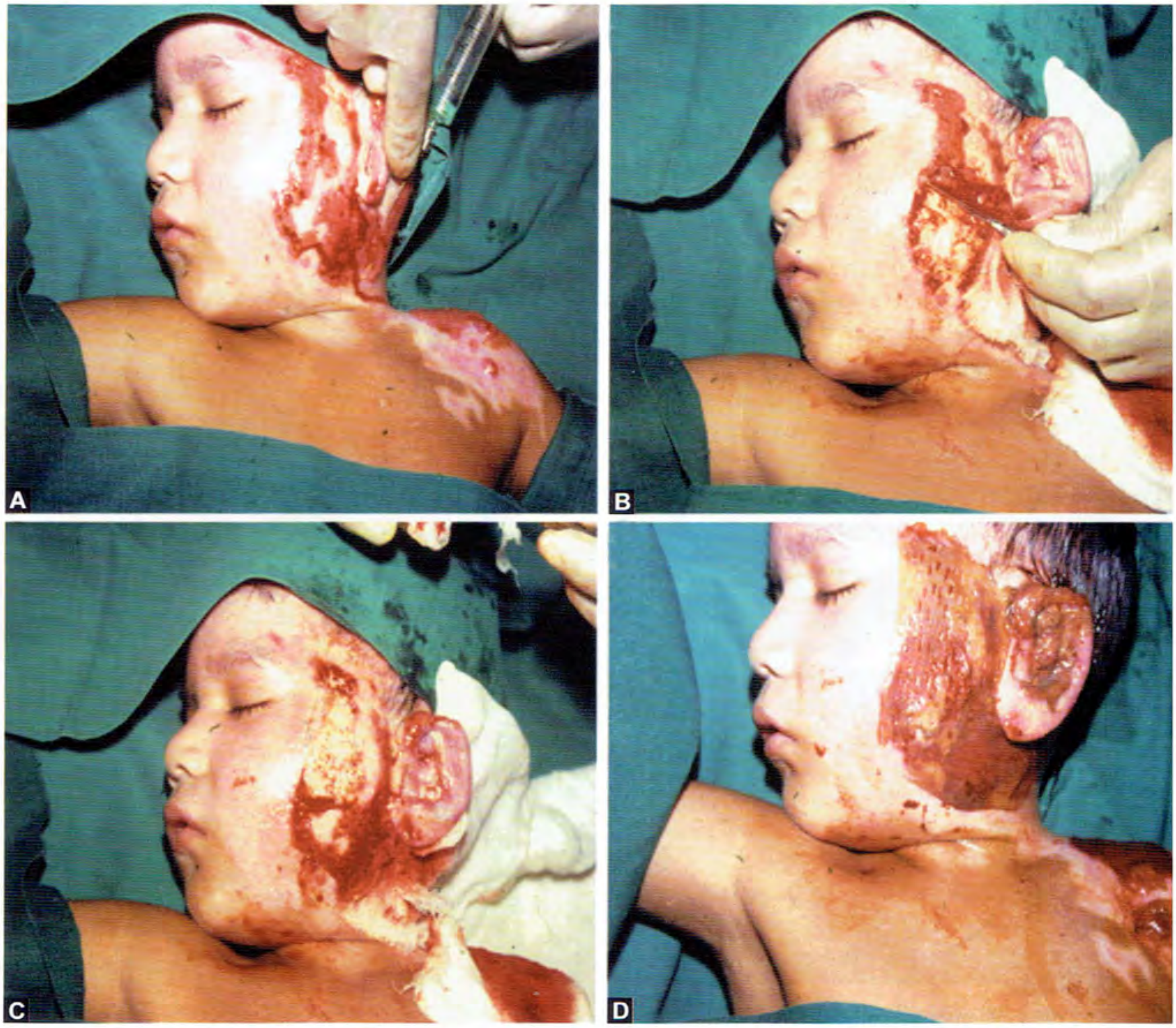


Fig. 5.7: Histological appearance of granulation tissue as seen under Microscope (40X) Note the pockets within the granulation tissue (shown by an arrow)



Figs 5.8A to D: Unhealthy granulation tissue. (A) Injection of saline-adrenaline 1:100,000 to prevent blood loss. (B) Debridement of granulation tissue. (C) Wound ready for skin grafting after debridement. (D) After complete healing

zone of stasis begins to recover in about 16 to 24 hours and circulation returns in few days post burn. When drying is prevented, oxygen consumption returns to this zone as early as 4th post burn day.

Winter (1963)[9] showed that the scab which forms over the exposed wound decreases the possibility of migration of keratinocytes ultimately causing impairment in the process of wound healing. On the other hand Zawaki (1974)[11] demonstrated that the occlusive dressings which do not allow the wound to dry also allow the tissue to retain the viability.

Lawrence (1975)[10]stressed that this perinecrotic z (Zone of stasis) can be saved by applying cover of suita dressing. If left without proper covering, the epithel elements in the dermis are lost due to dehydration. Zaw (1974)[11] supported this hypothesis. Janzekovic (197 demonstrated that early excision of the necrotic tissue a cover with skin graft improved the outcome in b patients.

Groves and Lawrence (1971) demonstrated significant improvement of the quality and quantity of der tissue in healed burns treated by tangential excision

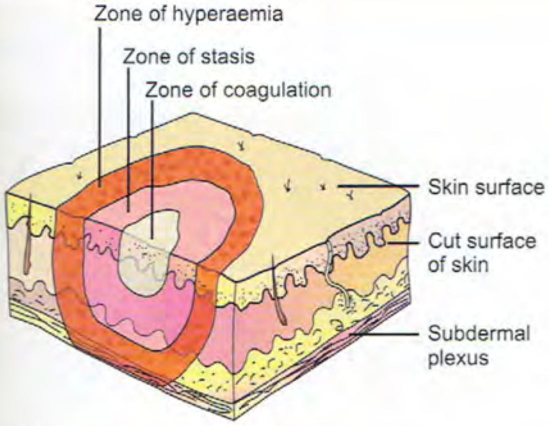


Fig. 5.9: Diagrammatic representation of three zones as described by Dr Douglas Jackson

autografting in comparison with vapor permeable material. The studies by Lawrence (1975)[10] have also demonstrated that a polyurethane film can prevent loss of epithelium in experimental tangentially excised burn wound.

Hinman and Maiback (1963)[13] showed that impermeable and semipermeable materials prevent the desiccation of the wound and indirectly help the process of healing (Fig. 5.10).

Keswani, Vartak and others (1990)[14] demonstrated conclusively that the use of moisture retaining dressing made from Boiled Potato Peel (BPP) did improve the quality of healing and shorten the period of healing. Compared with the treatment with the plain gauze, the application of potato peel dressing reduced or eliminated desiccation and hastened epithelial regeneration and promoted rapid healing. Pain relief after BPP Bandage was significant (Figs 5.11A to 5.12B).

The burn eschar is essentially a dried, devitalized skin covering the burn wound. Eschar does not provide the protection from infection. However, it is a good medium for growth of bacteria in the necrotic tissues. The property of the skin to prevent loss of fluids like plasma and its component is absent in eschar due to its high degree of permeability (Pruitt and Moncrief 1967).[15]

The necrotic tissue anywhere in the body allows bacterial colonization. The extensive burn itself is a large quantum of necrotic tissue on the body. The appreciation of this fact has led to the idea of removal of this tissue as early as early as possible (primary excision) with the hope that the septic complications can be avoided after removal of burned skin. This is expected to lead to a marked improvement in patient's condition and improved survival rate.

Hermans (1986)[16] reported the use of occlusive dressings using hydrocolloid material improved the quality of healing due to rapid re-epithelization, low incidence of infection and good cosmetic results. This led to better quality of life of the burn patient after healing. Lawrence and Lilly (1988) showed that hydrocolloid does act as a bacterial barrier.

Hermans (1992)[17] stressed that desiccation of the burn wound leads to the formation of the scab which decreases the migration of keratinocytes which eventually lead to impaired wound healing. On the other hand, the occlusive dressing with retention of moisture would not allow the

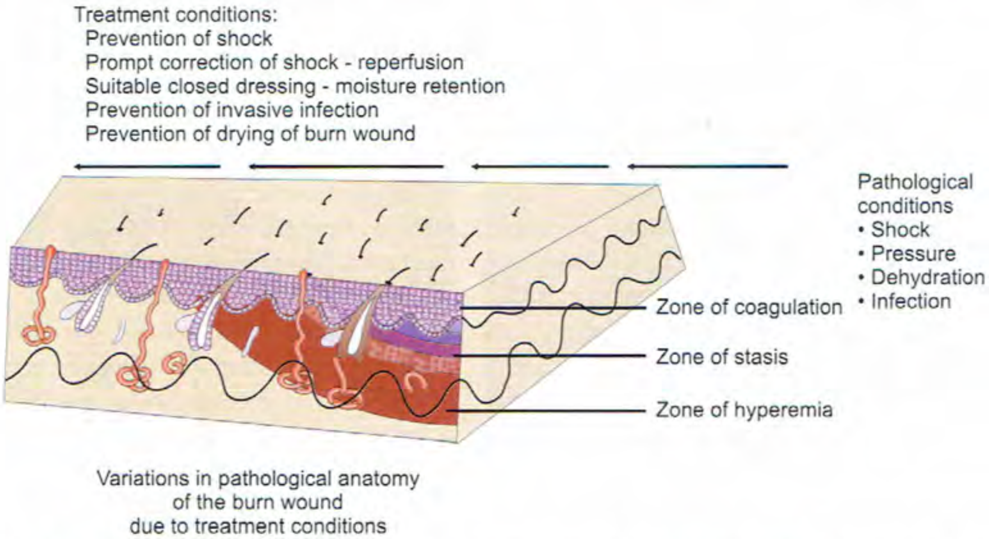
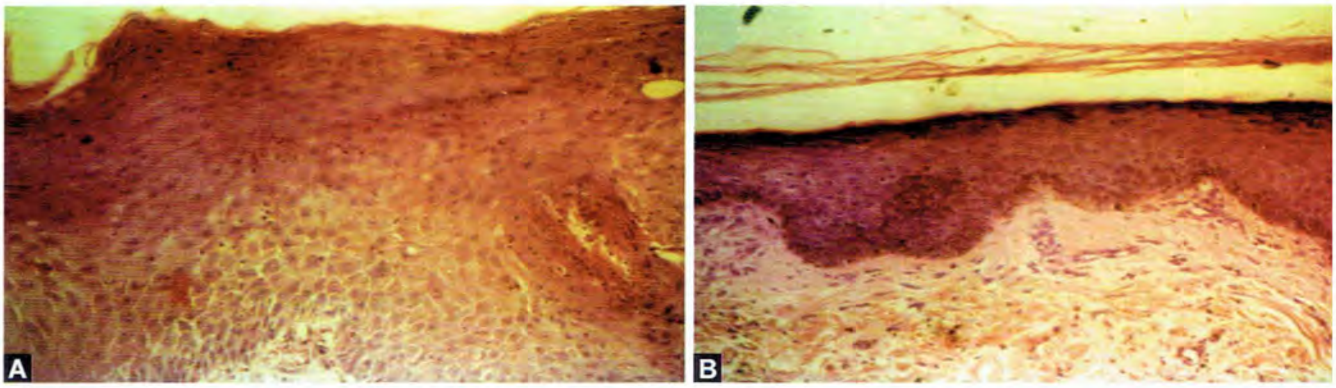
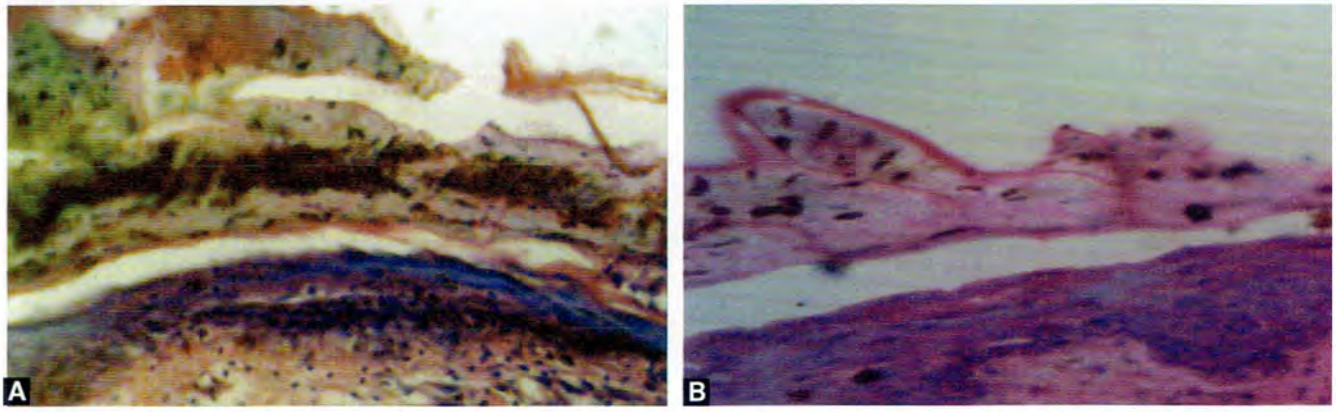

Treatment conditions:
Prevention of shock
Prompt correction of shock - reperfusion
Suitable closed dressing - moisture retention
Prevention of invasive infection
Prevention of drying of burn wound
Pathological conditions
• Shock
• Pressure
• Dehydration
• Infection
Zone of coagulation
Zone of stasis
Zone of hyperemia
Variations in pathological anatomy of the burn wound due to treatment conditions

Fig. 5.10: Changes in the burn wound which can occur due to various pathological conditions and as a result of response to adequate treatment



Figs 5.11A and B: Histological appearance (40X) after complete healing 21st post burn day. (A) Treated with BPP shows orderly stratificatio with basal layer of columnar epithelium. (B) Area treated with gauze and silver sulfadiazine cream shows complete healing with dysplasti stratification and basal layer of cuboidal cells



Figs 5.12A and B: Histological appearance (40X) 7th post burn day. (A) Shows the encrustation on the surface regeneration of the epidermi with dysplastic stratification of epidermis. (B) Section shows the moist slough on the surface following BPP dressing. Note more orderl stratification of the epidermis

wound to dry out and allows the tissues in the zone of stasis to retain the viability.

Semipermeable and impermeable dressings like synthetic films, 'Boiled Potato Peels', skin allografts or hydrocolloids prevent the desiccation of the wound and provide protection to the zone of stasis (Fig. 5.10).

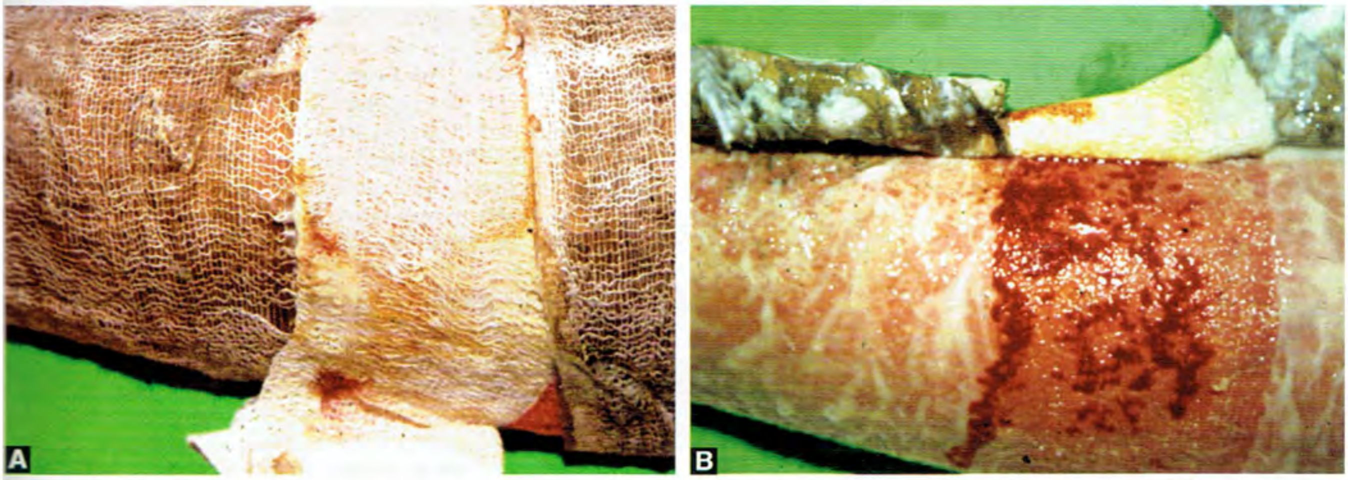
Some dressings (like cotton gauze) adhere to wound bed and cause damage to the newly growing epidermis and the granulation tissue. When dressing is removed during the dressing changes there is intense pain and bleeding from the wound bed. This clearly indicates that such dressings may cause a significant damage to the healing tissues (Figs 5.13A to 5.14D). Occlusive dressings, especially those which have the ability to retain moisture, do not adhere to the wound bed. There is no pain or bleeding during the dressing changes and thus indirectly help in faster healing of the burn wound.

When the dressing becomes dry and firmly adheres t the burn wound, destruction of growing neoepitheliur occurs during change of dressing and delays the proces of wound healing.

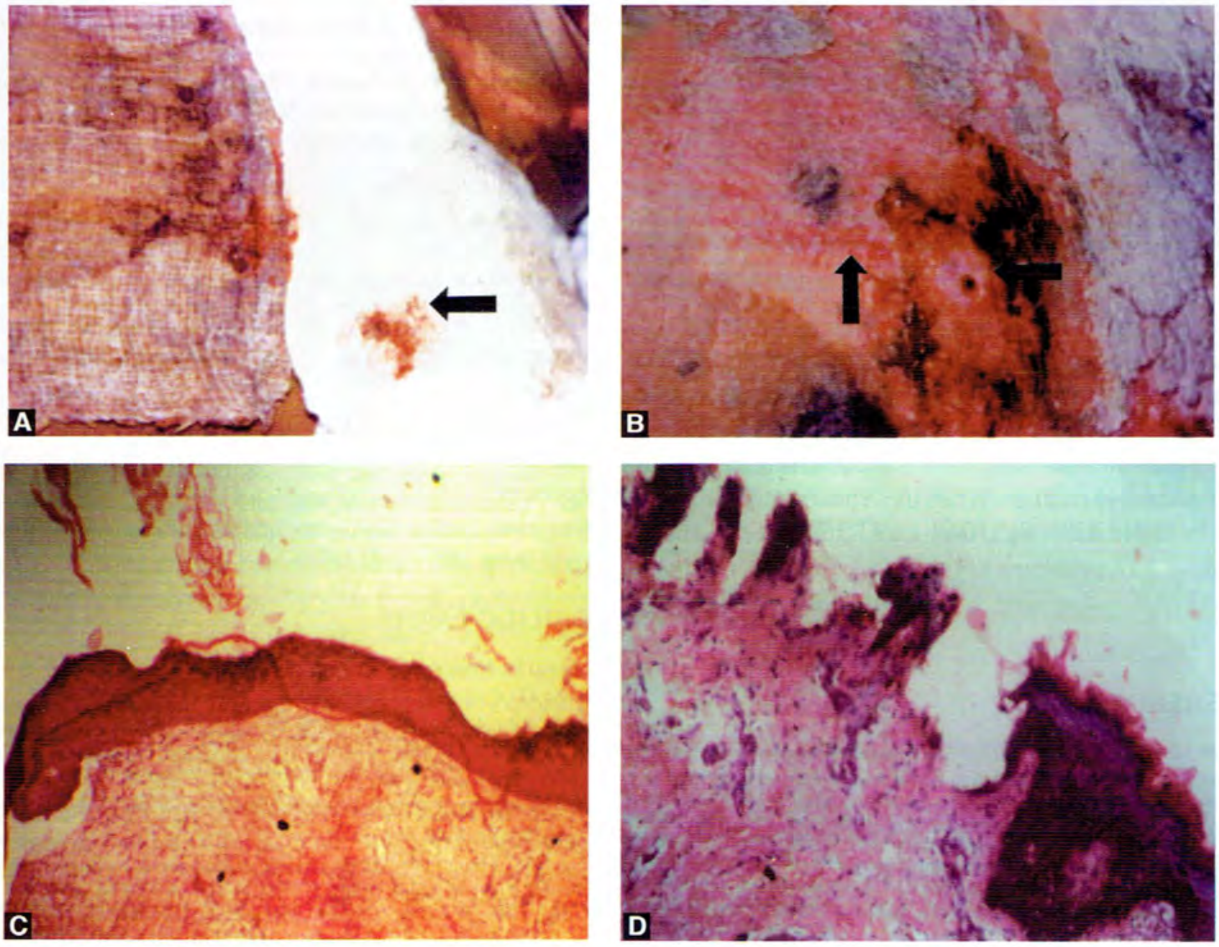
This epidermal damage is observed during the chang of dressing using gauze or similar materials. There is bleeding from the wound and also a severe pain indicating mechanical damage (Winter 1963).[9]

Hermans (1992)[17] has shown that the infection rate und the occlusive dressings is lower than that under th conventional dressing. *Pseudomonas aeruginosa* disappea in a moist wound environment and the wounds do heal i spite of bacterial contamination.

Lawrence and Lilly (1988)[18] have shown that dressing provide a physical barrier for microorganisms and again bacterial contamination. This helps reduce overall bacteri load which the host defense mechanisms have to deal wit

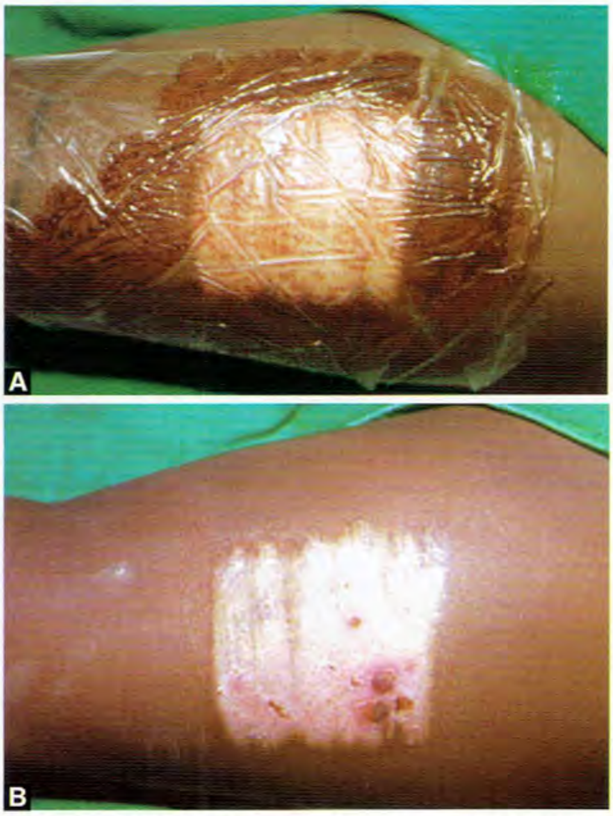


Figs 5.13A and B: (A) Burn injury treated with BPP bandage. Middle part of the wound treated with gauze. (B) Note areas of bleeding from the wound treated with gauze and resultant hemorrhage when the gauze dressing is removed, other areas are undamaged due to moisture retention and non-adherence



Figs 5.14A to D: (A) Burn injury on the chest right side treated with BPP dressing and left side treated with gauze. (B) Note that bleeding on left side after the dressing change (Horizontal arrow). (C) Complete healing of the epidermis can be seen on the side treated with BPP bandage. (D) Note the broken epidermis in the area treated with gauze

WOUND HEALING OF THE DONOR AREAS



Figs 5.15A and B: (A) Donor area of a split skin graft treated with cellophane paper. (B) Complete healing on 10th day

Donor areas also heal in the same manner as the burn wounds, i.e. by epithelial migration from the lining of epidermal appendages. Donor sites of the split skin graft are very painful, in the same way as superficial burns because of exposed nerve endings. When these donor sites are treated with occlusive dressings, they heal satisfactorily and do not cause any problems (Figs 5.15A and B). Occlusive dressing on the other hand, help rapid healing and significant relief of pain Hermans (1992)[17], Vartak (1991).[19]

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Chapter 6

Wound Healing in Burns

Mary Babu, N Shanmuganathan, B Karthikeya Prabhu

You were advised his flesh was capable of wounds and scars.

– William Shakespeare, Henry IV

INTRODUCTION

A burn injury is caused by damage to skin and other deeper tissues by extreme heat, flame and contact with heated objects or chemicals. Burn depth is generally categorized as first, second, or third degree. The treatment of burn wounds depends on the depth, extent and location of the burn as well as some additional factors. Treatment options may range from simply applying a cold pack immediately to skin grafts.

A first degree burn is superficial and has characteristics similar to a typical sun burn. The skin is red in color and sensation is intact. In fact, it is usually painful and damage is limited to the epidermis characterized by erythema and hyperemia. Second degree burns extend into the dermis to varying depths, involving papillary dermis alone or along with reticular dermis. In third degree burns, the damage progresses to the point of death of full thickness of skin. They are painless due to damage to nerve endings, exposing subcutaneous fat layer. Regardless of the type of burn, the result is fluid accumulation and inflammation in and around the wound. Moreover, it should be noted that the skin is the body's first defense against infection by microorganisms. Any damage to the skin predisposes a burn victim to infections not only at the site of the wound but also systemically. In addition to depth, the extent of the burn, i.e. the total body surface area (TBSA) burnt is also significant. This is measured in terms of percentage of TBSA burnt. Burns that cover more than 15% of the total body surface in adults can lead to hypovolemic shock requiring hospitalization for intravenous fluid resuscitation and wound care.

Location of burn on the body is even more important than the above factors as far as wound healing is concerned. In acute stage, burns of the face or signs of burns to the nose or mouth may require urgent guarding of the patient's airway, as edema can result in life-threatening obstruction of the airway. Burned tissue shrinks and can cause damage to underlying structures. Burns that extend circumferentially around limbs require their surgical release, escharotomy, to prevent gangrene of distal limb. Deep dermal burns heal with hypertrophic scarring while full thickness burns, if not split skin grafted lead to contractures.

FACTORS AFFECTING WOUND HEALING

There are several factors that affect the wound repair either beneficially or adversely. Any attempt to improve healing or prevent complications of healing should concentrate on the causes of the impairment.

Age

Age affects many aspects of wound repair. Several investigations have shown that rate of healing appears to slow down with increasing age. The fibroblast proliferation is at a slower rate in older individuals and they also have a decreased synthetic capability. The soluble collagen decreases and insoluble cross-linked collagen increases with age. The amount of elastin increases but the numbers of elastic fibers decrease to make skin less elastic. Possibly, in the earlier stages of life, wound healing is faster as compared to older individuals as it more closely resembles tissue regeneration seen in simpler animals.

Infection

The effects of infection on tissue repair can be divided into local or systemic effects. Several bacterial strains (*Pseudomonas, Staph. aureus*) have been found to impair wound healing. Bacterial contamination leads to an increased inflammatory response with release of more cytokines to attract more of the cells needed for wound repair. However, the wound becomes overwhelmed with virulent microorganisms and the bacterial proteases and the products of inflammatory cells lead to local tissue damage.

Nutrition

One of the major breakthroughs in the survival of burn patients has been made possible through aggressive nutritional support. Starting nutritional support as soon as possible after injury helps in preventing wound healing problems.

Vitamins and Trace Elements

Some vitamins are clearly important for normal tissue repair. Vitamin C is essential for normal collagen synthesis and deficiency leads to defects in connective tissue and impaired wound healing. Vitamin A induces fibroblasts to differentiate and to enhance collagen synthesis. Topical application of vitamin A may also improve healing. Vitamin E is a potent antioxidant and is likely to improve healing.

Deficiencies in trace elements can also lead to problems in wound healing. These trace elements are actually metals that are required for enzyme functions. Iron, Zinc, Copper, Manganese and Selenium are the most important trace metals during the process of healing as they are involved from the initial days of healing at different stages imparting specific functions.

Oxygen

Oxygen is so essential to healing and it is found that high levels of oxygen may be even more beneficial. The treatment of wounds with hyperbaric oxygen is based on this hypothesis. High levels of oxygen may lead to the formation of oxygen free radicals that have potential to damage tissues.

Steroids

The most notorious drugs associated with altered healin are steroids. Steroids are powerful inhibitors of th inflammatory response and therefore, have the potential t inhibit wound healing. Collagen synthesis and fibrosis ar inhibited with steroid use.

Chemotherapeutic Agents

They act by killing rapidly growing cells with the hope o eliminating proliferating cancer cells. However, the healin wound also requires cellular proliferation. It is reported tha most of the chemotherapeutic agents given, locally o systematically, impair healing.

Radiation

The impairment in wound healing seen after acute radiatio exposure is also due to inhibition of proliferating cells Radiation may cause chronic vasculitis that impairs loca blood supply and predisposes the patient to impaired healing

Local factors pertain mainly to persistence of debri within the wound, for example, devitalized tissues, clots foreign material including sutures and bacteria contamination. These can act as a physical barrier to th ordered development of granulation tissues and collage deposition or may exaggerate the evoked inflammator response. Ultimately, this can change the way the tissue heal, changing primary to secondary union or resolution t organization. Local tissue hypoxia, either chronic (e.g caused by radiation enteritis, atherosclerosis or diabeti microvascular disease) or more acute (e.g. caused b vascular damage or tight sutures) may compromise healing Mobile wounds do heal but more slowly than those hel together by sutures.

Patients with uncontrolled diabetes suffer from a variet of problems in healing. High blood sugar levels decreas both neutrophil chemotaxis and phagocytic ability Additionally, a sugar-rich environment favors microbia growth, further interfering with healing.

WOUND HEALING: GENERAL

A wound is a break in the epithelial integrity of the skin an may be accompanied by disruption of the structure an function of underlying normal tissues. A wound may resul from precise disruption of tissue by the surgeon's knif (incision) or there may be widespread damage of tissues (e.g. after major musculoskeletal trauma and burns). A wound may also result from a contusion, laceration or an abrasion. The continuity of the skin must be restored expeditiously as it plays a crucial role in maintaining homeostasis.

Wound healing is a highly complex physiological process. It involves the interplay of various cellular and biochemical components, which drive the repair cascade. In addition, several enzymatic pathways also become active during repair and help the tissues to heal. Following tissue injury, blood vessels are disrupted resulting in extravasation of blood components. This is followed by platelet aggregation and blood coagulation resulting in the generation of a fibrin-rich clot that fills the discontinuity in the injured tissue and re-establishes hemostasis. Subsequently, the migration of inflammatory cells to the wound site begins and these cells debride the wound. This marks the beginning of the inflammatory phase of wound healing. This is followed by the formation of new blood vessels or angiogenesis within the newly forming granulation tissue. Fibroblasts deposit and remodel the granulation tissue. Granulation tissue is then transformed to the final product of the healing process, a scar.

Although the desirable final result of coordinated healing would be the formation of tissues with a similar structure and comparable functions as of an intact skin, regeneration is uncommon (with notable exceptions such as early fetal healing). Healing, however, does result in a structurally and functionally satisfactory but not identical outcome. Wound healing processes seem to be strictly regulated by multiple growth factors and cytokines released at the wound site. Alterations that disrupt controlled timely healing processes extend tissue damage and prolong repair.

WOUND HEALING: TYPES

Primary healing (healing by first intention) occurs when a wound is closed within 12-24 hours of its creation (e.g. clean surgical incision, clean laceration). The wound edges are approximated directly using sutures, tissue glue, tapes or a mechanical device. Because of an appropriate balance between all phases of the healing process (including cellular proliferation, collagen metabolism, activity of matrix metalloproteinase and degradation of the extracellular matrix), the wounds heal well and proceed rapidly towards complete closure.

Secondary healing (healing by second intention) occurs in a wound with extensive loss of soft tissues, as seen in major trauma, severe burns and after some surgical procedures (e.g. laparotomy). Regeneration of epithelial cells alone cannot restore the original architecture. There is ingrowth of granulation tissue from the wound margin, followed by accumulation of extracellular matrix with the laying down of collagen (Fig. 6.1). These open, full-thickness wounds thus close by subsequent wound contraction and epithelialization. For example, large defects

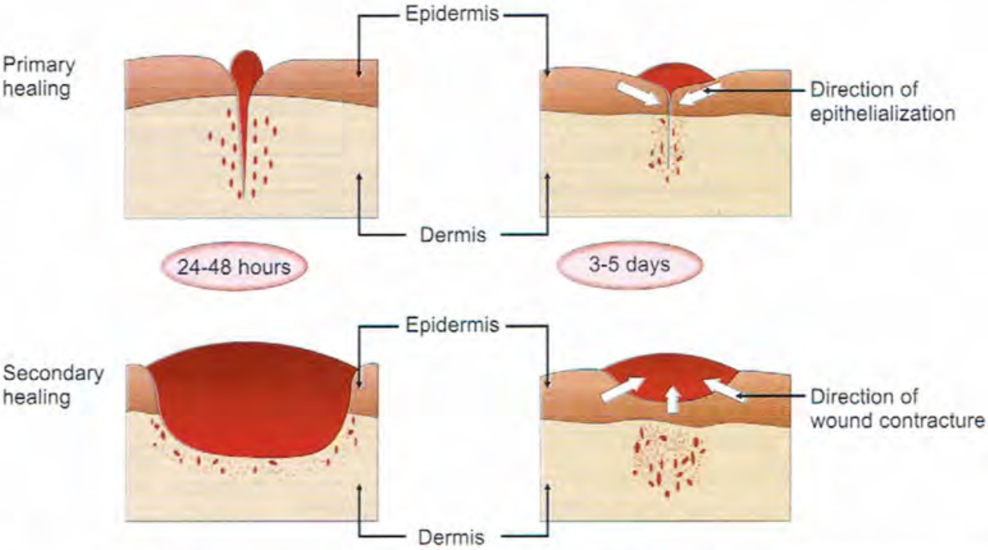


Fig. 6.1: Primary and secondary healing. *Adapted from*: Pathophysiology of wound healing. Surgery (Oxford), Volume 20, Issue 5, 2002, Pages i-v CM Halloran and JP Slavin

of skin may reduce to 5-10% of their original size within six weeks, largely by contraction. Myofibroblasts, having structural properties between those of a fibroblast and a smooth muscle cell, are thought to play a key role in this type of healing. They appear in the wound about three days after wounding and increase in number to a maximal level between 10 and 21days. Healing by secondary intention is slower, may lead to contractures (particularly over joints) and may lead to functional restriction.

Healing of partial-thickness wounds is seen in injuries such as superficial dermal burns, donor sites of split-thickness skin grafts and abrasions where the injury involves the epidermis and the papillary part of the dermis. Healing occurs purely by epithelialization from the epidermal appendages and anatomical and physiological restoration is virtually complete. Healing of some very deep dermal and full thickness burns require skin grafting or else a contracture forms.

PHASES IN WOUND HEALING

Inflammatory Phase

Inflammation is a highly effective component of the innate initial reaction of the body to injury. It is an important consequence of injury and one that normally leads to tissue repair and restoration of function. The inflammatory response can be subdivided into vascular and cellular responses. Early in the wounding process, local vasodilatation, blood and fluid extravasation into the extravascular space and blocking of lymphatic drainage can produce cardinal signs of inflammation, including redness, swelling and heat. This acute inflammatory response usually lasts between 24 and 48 hours and may persist for up to 2 weeks in some cases. Tissue injury causes blood vessel disruption and bleeding. Platelets adhere, aggregate and release many mediators to facilitate coagulation. Although hemostasis is the major function of blood coagulation, a secondary but equally important function of platelets is to initiate the healing cascade via release of chemo-attractants and growth factors. At the same time, the clot provides a matrix scaffold for the recruitment of cells to an injured area. In response to these important mediators, leukocytes, including neutrophils and macrophages infiltrate the wounded area and assist in cleaning and removing damaged tissue debris and foreign particles. Once in the wound site, activated macrophages release several important growth factors and cytokines, initiating granulation tissue formation (Fig 6.2).

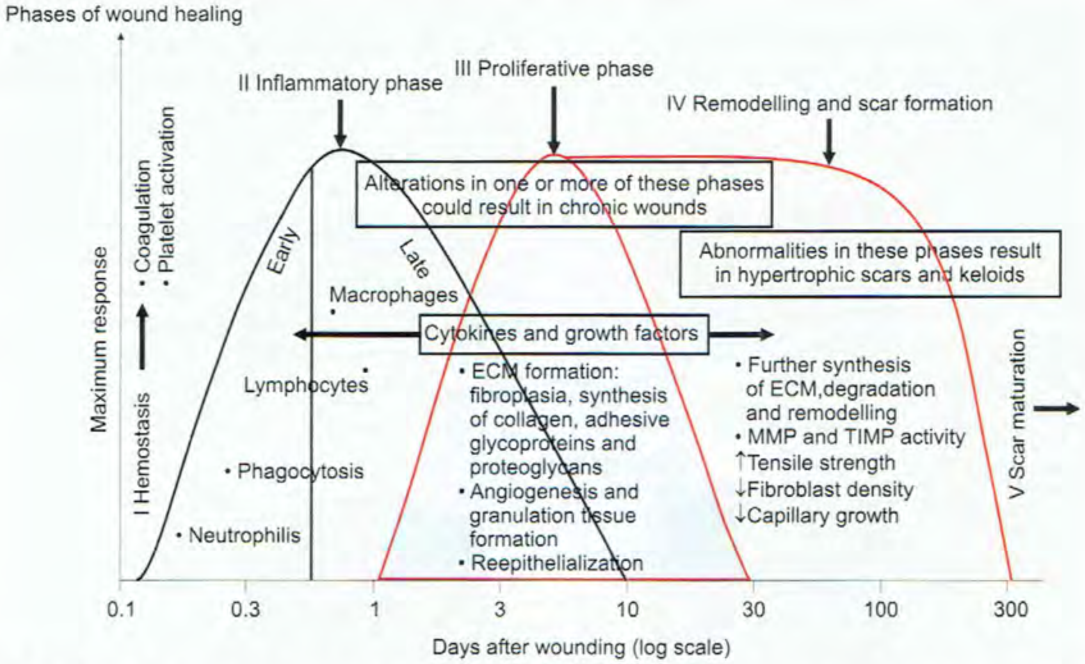


Fig. 6.2: ECM: Extracellular matrix; MMP: Metalloproteinases; TIMP: Tissue inhibitors of metalloproteinases. *Adapted from*: Clark RA. In: Goldsmith LA, (ed.) Physiology, biochemistry, and molecular biology of the skin, 2nd edn, Vol. I. New York: Oxford University Press, 1991, p 577

The Cellular Response and Inflammation

The cellular response of the inflammatory phase is characterized by the influx of leukocytes into the area of injury. In the early inflammatory state, neutrophils and monocytes are the predominant cells at the site of injury. Shortly after injury, neutrophils and monocytes begin to emigrate from capillaries into the wounded tissue with neutrophils being the first to arrive in great numbers. Later in inflammation, the number of neutrophils decline and macrophages (tissue-derived monocytes) predominate. Neutrophils and monocytes are recruited to the wound by chemotactic factors released during hemostasis and by mast cells. Chemotactic factors generated during the coagulation process, such as kallikrein, fibrinopeptides released from fibrinogen and fibrin degradation products, also serve to up-regulate the expression of important intercellular adhesion molecules. Substances released by mast cells, such as tumor necrosis factor, histamine, proteases, leukotrienes (LTs), and cytokines (interleukins), represent additional sources of chemotactic signals for the recruitment of leukocytes. Growth factors like PDGF and Transforming Growth Factor-β (TGF-β) are also potent chemotactic factors for leukocytes. Once in the wound site, integrin receptors found on the cell surface of neutrophils enhance cell-matrix interactions. This allows neutrophils to perform their function of killing and phagocytosing bacteria and damaged matrix proteins within the wound bed. Neutrophil infiltration normally lasts for only a few days but the presence of wound contamination prolongs the presence of neutrophils within the wound and may delay healing. Monocytes immigrate into tissue spaces and transform into larger phagocytic macrophages that soon become the predominant cell type during the latter part of the inflammatory phase. Monocytes are initially attracted to the wound site by some of the same chemo-attractants that attract neutrophils and their recruitment continues through signals released by monocyte-specific chemo-attractants such as monocyte chemo-attractant protein-1 and macrophage inflammatory protein-1. The extracellular matrix degradation products, collagen fragments, fibronectin fragments and thrombin are also specific chemo-attractants for monocytes. Macrophages are considered to be the most important regulatory cells in the inflammatory reaction. Macrophages phagocytose, digest and kill pathogenic organisms, scavenge tissue debris and destroy any remaining neutrophils. After binding to the extracellular membrane, bacterial, cellular and tissue phagocytoses and subsequent destruction are accomplished through the release of biologically active oxygen intermediates and enzymatic proteins.

Chemical Mediators of Inflammation

A number of chemical substances are involved in the initiation and control of inflammation. These chemicals work in concert: some are protagonists and others are antagonists of inflammation. They can be grouped as vasoactive amines of histamine and serotonin, plasma proteases of kinins and complements, plasma proteins of the coagulation system, arachidonic acid metabolites of prostaglandins (PGs) and LTs, cytokines and growth factors and free radicals of nitric oxide and oxygen-derived free radicals (Fig. 6.3).

Proliferative Phase

The initial inflammatory responses to injury provide the necessary framework to the subsequent production of a new functional barrier. In this phase of healing, cellular activity predominates. The major events during this phase are the creation of a permeability barrier (i.e., re-epithelialization), the establishment of appropriate blood supply (i.e. angiogenesis), and reinforcement of the injured dermal tissue (i.e., fibroplasia).

Reepithelialization (Fig. 6.4)

Reepithelialization is the process of restoring an intact epidermis after cutaneous injury. It generally involves several processes, including the migration of adjacent epidermal keratinocytes into the wound, the proliferation of keratinocytes used for the supplement of the advancing and migrating epithelial tongue, the differentiation of the neoepithelium into a stratified epidermis, and the restoration of an intact basement membrane zone (BMZ) that connects the epidermis and the underlying dermis.

Keratinocyte Migration

Keratinocyte migration is an early event in wound reepithelialization. The keratinocytes initially respond to an epidermal defect by migrating from the free edges of the wound within 24 hours. The keratinocyte migration in partial-thickness wounds also occurs from remaining skin appendages and wound edges. Epidermal stem cells from the hair follicle are now thought to originate from the hair bulb that is believed to be the germinative portion of the hair and serve as a reservoir for keratinocytes in wound healing.

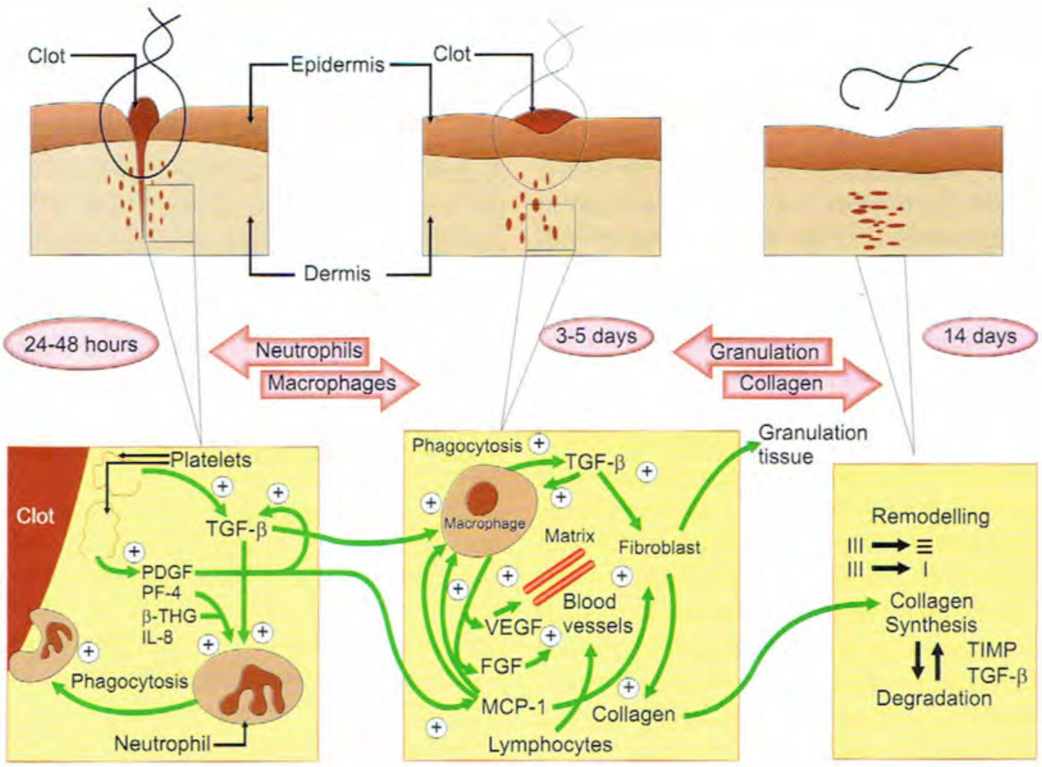


Fig. 6.3: Cytokines and healing. *Adapted from*: Pathophysiology of wound healing. Surgery (Oxford), Volume 20, Issue 5, 2002, Pages i-v CM Halloran and JP Slavin

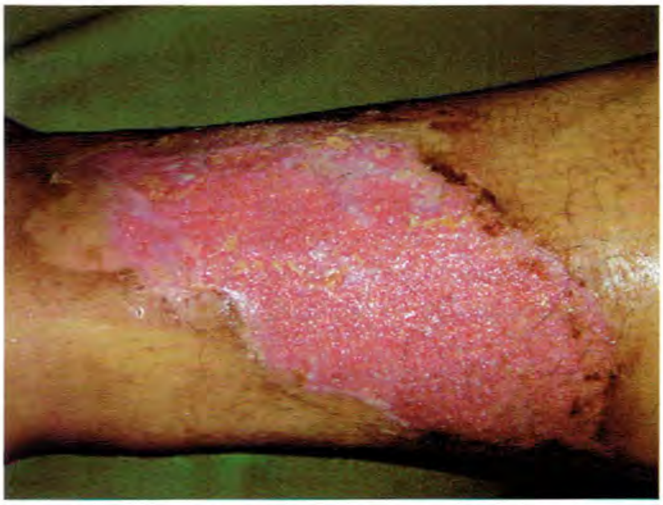


Fig. 6.4: Superficial dermal burns on lower leg showing reepithelialization. The epithelial buds can be seen over the surface

A number of processes must occur within keratinocytes in preparation for migration. Approximately 12 hours after wounding, a series of events, such as flattening and elongation of keratinocytes, development of pseudopod-like projections of lamellipodia, loss of cell-cell and cell-matrix contacts, retraction of intracellular tonofilaments and formation of actin filaments at the edge of their cytoplasm, occurs. Although keratinocytes are migrating, their proliferative potential is inhibited. Migrating basal keratinocytes may express selective cell surface markers such as CD44 and some markers usually expressed by squamous cells.

Several elements have been implicated in keratinocyte migration, including the extracellular matrix, integrin receptors, matrix metalloproteinases (MMPs) and growth factors. An early provisional matrix formed by fibrin, fibronectin, and type V collagen enables keratinocytes to migrate and dissect under eschar and debris that may be covering the wound. Keratinocytes use their surface integrin receptors to interact with a fibronectin-rich provisional matrix. The direction of migration is also regulated by the binding of keratinocytes to integrin receptors on the newly formed collagen molecules in the wound bed. Dissociation of this binding allows the keratinocytes to migrate forward. Matrix metalloproteinases also play an important role in keratinocyte migration by their involvement in this dissociation. Migrating keratinocytes produce MMPs, such as MMP-9, which specifically degrades type IV collagen and laminins in the basement membrane. This allows cells to leave the basement membrane and migrate into the wound. Matrix metalloproteinase-1 disrupts any attachment to a fibrillar collagen and facilitates the continued migration of keratinocytes in the wound.

Keratinocyte Proliferation

Reepithelialization also involves increased proliferation of keratinocytes located near the cells of the migrating front tongue. This proliferating source of keratinocytes ensures an adequate supply of cells to migrate and cover the wound. When migration ceases, possibly as a result of contact inhibition, keratinocytes reattach themselves to the underlying substratum, reconstitute the basement membrane and then resume the process of terminal differentiation to generate a stratified epidermis. One can observe that there are single-layered keratinocytes toward the wound center whereas there are multiple-layered or stratified keratinocytes near the wound edges. Differentiation then follows proliferation as the proliferative index is found significantly increased at the wound center, whereas the differentiation of the neoepidermis (keratins 1/10, filaggrin, and loricrin) and regeneration of the dermoepidermal junction (laminin 5 and collagen IV) are more advanced toward the wound margin. Epidermal growth factor (EGF), keratinocyte growth factor and Transforming Growth Factor-α (TGF-α) have been shown to be among the important stimuli for keratinocyte migration, proliferation, and reepithelialization.

Restoration of the Basement Membrane Zone (BMZ)

The formation of an intact BMZ between the epidermis and the dermis is essential for the reestablishment of skin integrity and function. Within 7 to 9 days after reepithelialization, the BMZ reforms. The BMZ forms an adhesive structure because its superior aspects serve as an attachment site for basal keratinocytes through the formation of a hemidesmosome-anchoring filament complex whereas the inferior or lower portion stabilizes the attachment to the underlying dermis by anchoring fibrils. The importance of BMZ is evidenced by a group of inherited blistering diseases known as epidermolysis bullosa. In these conditions, mutations of one of a variety of BMZ proteins, such as hemidesmosome collagen XVII in atrophic epidermolysis bullosa, defects of laminin 5 of a major anchoring filament in junctional epidermolysis bullosa and deficiency of collagen VII anchoring fibrils in dystrophic epidermolysis bullosa lead to blistering and ulcer formation.

The skin BMZ consists of many extracellular matrix proteins; among these, collagens and laminins are the major components. Collagen IV is the most abundant component and forms a 3-dimensional lattice network within the electron-dense or lamina densa portion of skin BMZ. Collagen VII proteins, also called anchoring fibrils, span from the lamina densa to the upper papillary dermis, where they form a structure known as an anchoring plaque. Another important collagen is collagen XVII, also known as bullous pemphigoid antigen (bullous pemphigoid antigen 2 or bullous pemphigoid antigen 180), which is a 180-kDa transmembrane protein located on the hemidesmosome complex of basal keratinocytes. Laminins are the major noncollagenous extracellular matrix components in a wide range of BMZs within human tissues. Several laminins are present in the BMZ of the dermoepidermal junction. Composed of a, b and c subunits, laminin 5 (a3b3c2) and laminin 10 (a5b1c1) are the major laminins in skin BMZ and found to be actively involved in wound repair. In response to wounding, keratinocytes in the migrating front edge deposit laminin 5; this serves as a track to allow subsequent keratinocytes to migrate. Recently, a new laminin member, laminin 10, has been described; it is located within the lamina densa. Laminin 10 was also found to be a major laminin of dermal microvascular blood vessels. Laminin 10 knockout mouse skin was found to exhibit discontinuity in BMZ lamina densa and defects in hair development.

Reconstitution of the Dermis

Dermal reconstitution begins approximately 3 to 4 days after injury, characterized clinically by granulation tissue formation, which includes new blood vessel formation or angiogenesis and the accumulation of fibroblasts and ground matrices, named fibroplasia. The provisional extracellular matrix that is formed in part by the fibrin clot, which is rich in fibronectin, promotes granulation tissue formation by providing scaffolding and contact guidance for cells to migrate into wound spaces and for angiogenesis and fibroplasia to occur in an effort to replace the wounded dermal tissue.

Fibroplasia

Fibroplasia describes a process of fibroblast proliferation, migration into wound fibrin clot and production of new collagen and other matrix proteins, which contribute to the formation of granulation tissue. As an early response to injury, fibroblasts in the wound edges begin to proliferate and by approximately day 4 start to migrate into the provisional matrix of the wound clot, where they lay down a collagen-rich matrix, including collagens, proteoglycans, and elastin. Once the fibroblasts have migrated into the wound, they gradually change to profibrotic phenotypes and switch their major function to protein synthesis. Fibroblasts are also modulated into phenotypes of myofibroblasts and participate in wound contraction.

Structural molecules of the early extracellular matrix, such as fibronectin and collagen, contribute to granulation tissue formation by providing a scaffold for contact guidance and a reservoir for cytokines and growth factors. Fibronectin, a glycoprotein, is a major component of the gel like substance initially secreted and provides for enhanced fibroblast activity. Fibronectin allows fibroblasts to bind to the extracellular matrix and provides an adherent base for cell migration. The fibronectin matrix also provides a scaffold for collagen fibrils and mediates wound contraction. Major fibroblastic chemotactic factors are, in part, derived from macrophages present in the wound. Both PDGF and TGF-β can stimulate fibroblast migration and up-regulate the expression of integrin receptors. Epidermal growth factor and fibroblast growth factor, among others, modulate fibroblast proliferation and migration. Fibroblast proliferation is also stimulated by an acidic low-oxygen condition found in the center of the wound. As angiogenesis proceeds with the formation of new vessels and increased oxygen carrying capacity, this stimulus diminishes.

Angiogenesis

Angiogenesis refers to new vessel growth by the sprouting of preexisting vessels adjacent to the wound. As in most normal adult tissues, dermal blood vasculatures remain quiescent. In response to the injury, microvascular endothelial cells initiate an angiogenic process consisting of activation of endothelial cells. Newly formed vessels participate in granulation tissue formation and provide nutrition and oxygen to growing tissues. In addition, inflammatory cells require the interaction with blood vessels to enter the site of injury. During angiogenesis, endothelial cells also produce and secrete biologically active substances or cytokines.

Angiogenesis involves a phenotypic alteration of endothelial cells, directed migration and various mitogenic stimuli. Cytokines released by macrophages stimulate angiogenesis during wound healing, as do low-oxygen tension, lactic acid and biogenic amines produced in the wound. Several growth factors have been shown to play critical roles in wound angiogenesis, including vascular endothelial growth factor, angiopoietins, fibroblast growth factor and TGF-β. Vascular endothelial growth factor, also known as vascular permeability factor, which exerts its biologic activity predominantly on endothelial cells, is a key mediator of angiogenesis.

Formation of the Extracellular Matrix (ECM)

Besides providing turgor to soft tissues and rigidity to bone, extracellular matrix supplies a substratum for cell adhesion and critically regulates the growth, movement and differentiation of the cells within it. The extracellular matrix consists of fibrous structural proteins (collagens, elastin) and an interstitial matrix composed of adhesive glycoproteins embedded in a proteoglycan and glycosaminoglycan gel. The seemingly random array of interstitial matrix in connective tissues soon becomes highly organized around epithelial cells, endothelial cells and smooth muscle cells, forming the specialized basement membrane (which directs cell polarity and is required for the orderly renewal of the epithelial tissue).

Collagens are synthesized by fibroblasts and are the most abundant family of proteins in the body. They provide strength and integrity to all tissues and so play a vital role in wound repair. Platelet-derived growth factor, basic fibroblast growth factor, transforming growth factor-β, interleukin-1, tumor necrosis factor (TNF) induce collagen synthesis during the proliferative and remodeling phases. Collagens are composed of three protein α-chains braided into a rope-like triple helix; the individual chains are able to intertwine tightly because each α polypeptide has one glycine molecule at every third position. More than 30 distinct α-chains form about 20 different collagen types (some of which may be unique to specific cells and tissues). Some collagen types (e.g. I, III, V) form fibrils due to lateral crosslinking of the triple helices and form most of the connective tissue in healing wounds; other collagens (e.g. type IV) are non-fibrillar and become components of the basement membrane.

Adhesive glycoproteins are structurally diverse proteins that link the components of the extracellular matrix to one another and to cells and include fibronectin, laminin and thrombospondin.

- Fibronectin, a large (400-kD) disulphide-linked heterodimer, is associated with cell surfaces, basement membranes and the pericellular matrix. It has specific domains that bind to a wide spectrum of components of the extracellular matrix (e.g. collagen, fibrin and proteoglycans) and can also attach to cell integrins. Adhesive matrix proteins such as fibronectin can directly mediate the attachment, spreading and migration of cells. Fibronectin also enhances the sensitivity of certain cells (e.g. endothelial) to the proliferative effect of growth factors by activating intracellular signaling pathways.
- Integrins are a family of α/β heterodimeric glycoproteins that mediate cell-cell and cell-matrix adhesion. They are important in the integration of function between the extracellular matrix and the cytoskeleton.

Proteoglycans consist of glycosaminoglycans (e.g. dermatan sulfate, heparan sulfate) linked to a protein backbone; they help to regulate the structure and permeability of the extracellular matrix. Proteoglycans can modulate the growth and differentiation of cells (e.g. syndecan binds extracellular matrix collagen, fibronectin, thrombospondin and basic fibroblast growth factor and associates with the intracellular actin cytoskeleton to maintain the normal morphology of epithelial sheets). Glycosaminoglycans without a protein core (e.g. hyaluronan) are important constituents of the extracellular matrix.

Formation of granulation tissue in 3-5 days (indicative of optimal healing) is well established. Granulation tissue has a pink, soft, granular gross appearance (such as that seen beneath the scab of a skin wound). Histologically, it is denoted by proliferating fibroblasts and loops of capillaries in a loose extracellular matrix. This phase is characterized by angiogenesis or formation of new blood vessels from pre-existing vessels at the site of injury (neovascularization).

Remodeling Phase

Remodeling consists of the deposition of the matrix and its subsequent changes over time. It occurs throughout the entire wound repair process as fibrin clot formed in the early inflammatory phase is replaced by the granulation tissue that is rich in type III collagen and blood vessels during the proliferative phase and subsequently replaced by a collagenous scar predominantly of type I collagen with much less mature blood vessels. One of the characteristics of wound remodeling is the change of extracellular matrix composition. Collagen fibers constitute approximately 80% of the dry weight of normal human dermis and are the principal proteins providing structure, strength and stiffness to dermal tissue. In healthy adults, type I collagen accounts for approximately 80% of collagens and type III collagen constitutes 10% of collagens in the dermis. During early wound healing, however, similar to the fetal dermis, type III collagen is the predominant collagen synthesized by fibroblasts in granulation tissue. Type III collagen first appears after 48 to 72 hours and is maximally secreted between 5 and 7 days.

The total amount of collagen increases early in repair, reaching a maximum between 2 and 3 weeks after injury. Over a period of 1 year or longer, the dermis gradually returns to the stable preinjury phenotype, consisting largely of type I collagen. Tensile strength, a functional assessment of collagen, increases to 40% of strength before the injury at 1 month and may continue to increase for 1 year, reaching up to 70% of its preinjury strength. With wound closure, a gradual turnover of collagen occurs as type III collagen undergoes degradation and type I collagen synthesis increases. The process of this conversion of the dermis is accomplished through a tightly controlled synthesis of new collagen and lysis of old collagen, mainly carried out by the actions of MMPs. The stimulus for this conversion may be the biomechanical stress and strain placed across a closed wound. Matrix metalloproteinases are usually not detectable or at very low levels in healthy resting tissue and are instead induced during wound repair, in response to cytokines, growth factors, and/or cell contact with the extracellular matrix. The catalytic activity of MMPs is also controlled in part by a family of tissue inhibitors of metalloproteinases. Tissue inhibitors of metalloproteinases specifically bind MMPs and are their natural inhibitors. The balance between the activities of MMPs and tissue inhibitors of metalloproteinases is also critical to wound repair and remodeling.

The final phase of wound healing is resolution. It involves remodeling of ECM and contraction of the wound. Contraction of the wound occurs after formation of the granulation tissue and requires the fibroblasts to assume a myofibroblast phenotype. Remodeling of the ECM, which leads to the conversion of granulation tissue to scar tissue, follows this and is dependent on the continual synthesis and degradation of collagen fibrils. Metalloproteinases participate in regulating mechanisms in all of these repair processes. For example, inflammation is shaped by cytokines and chemokines, which arise largely from resident cells (epithelium, endothelium, fibroblasts, etc.). MMPs can activate these mediators, by cleaving them from the cell surface or processing them to increase their activity or degrade them, thereby inhibiting inflammatory signals. Also the MMPs are able to cleave components of cell–cell junctions and cell–matrix contacts within the epithelium to promote reepithelialization. Furthermore, MMPs are involved in remodeling the scar ECM either directly by proteolytic degradation of proteins, such as collagens or indirectly via their ability to affect cell behavior. Alteration of the ECM is integral to the resolution of wound healing but also has implications in regulation of inflammation. Thus, MMPs are key regulators of multiple aspects of tissue repair and further study of these enzymes and their interaction with their substrates will not only advance our basic knowledge of wound healing, but also provide insight into possible therapies.

FETAL VS ADULT

Wound healing is a complex process involving cellular migration, proliferation and differentiation with extracellular matrix (ECM) synthesis and matrix remodeling. Many of these mechanisms are common to development and wound healing. Recent studies have demonstrated significant differences between adult and fetal wound healing. In a fetus, healing occurs rapidly by a regenerative process without scar formation while in an adult it leads to scar formation, often limiting tissue function and restricting further growth. The fetal response to a wound may be related to its developmental stage, the sterile aqueous fetal environment or a combination of both factors. For the interstitial collagens (types I, III, V, and VI), the critical difference between fetus and adult is in the supramolecular organization of the collagen fibrils deposited in the wound. In the fetus, a reticular collagen pattern is restored and this is indistinguishable from the surrounding normal tissue. In the adult, large, parallel bundles of collagen, typical of scar tissue, are seen running across the wound site approximately at right angles to the basement membrane, thus disrupting normal tissue architecture. One fundamental difference between fetal and adult wound healing is, therefore, the control and patterning of collagen fibrillogenesis. Collagen fibril formation is a complex process which is only partially understood. Collagen types I, III and V are present within the same fibrils and interactions between different collagens will alter fibril size *in vitro*. Interactions with other macromolecules are also likely to influence collagen fibrillogenesis. Proteoglycans will alter fibril size as will fibronectin. Finally the role of the cell has to be considered. Fibroblasts separate stages of fibril formation in distinct cellular compartments allowing the cell to control the final fiber orientation. Several of the differences in the ECM of fetal and adult wounds, shown in the studies, potentially may be altering collagen fibril formation. Collagen types I, III, V and VI are present concurrently in both fetal and adult wounds but the relative proportions of these collagen types are unknown. Fetal tissues contain a higher proportion of type III collagen to type I collagen and this may influence collagen fibril size. The role of glycosaminoglycans is intriguing as studies have shown high levels of hyaluronic acid in fetal wounds whilst addition of hyaluronic acid to an adult tympanic membrane wound decreased scar formation and altered the degree of organization of collagen within the wound. The interactions of sulfated GAGs with collagen may alter fibril formation within the fetal wound. During development, fetal bovine and chick collagen fibrils were small in diameter at a time when hyaluronic acid was abundant and a rapid increase in collagen fibril size during mid gestation coincided with a decrease in hyaluronic acid and increase in chondroitin sulfate content. The different patterning of collagen fibrils in healing fetal (regeneration of normal dermal pattern) and adult (scar consisting of parallel, densely packed collagen fibrils at right angles to the epithelial surface) wounds may therefore relate to differences in the cellular and extracellular milieu influencing the process of fibrillar differentiation in the primary wound 'scaffolding' into which fibroblasts migrate and lay down collagen fibrils. The fetal wound shows rapid epithelial and mesenchymal migration, the latter into a loose honeycomb lattice, rich in (and presumably held open by) glycosaminoglyeans, proteoglycans and glycoproteins. Thus, fetal fibroblasts can rapidly and easily migrate into a loose wound lattice and rapidly deposit collagen fibers. The transmission of mechanical forces to these fibrils by fibroblast tractioning, within a compliant matrix, may orientate the fibrils in a reticular pattern similar or identical to that of the normal dermis. By contrast, adult wounds show slow fibroblast migration into a denser, more resistant, wound matrix where fibroblasts seem to migrate more easily up and down the wound margins rather than into the body of the wound scaffold itself. Within the adult wound the 'stiffer' matrix and the forces of wound contracture generated by myofibroblasts may alter the orientation of collagen fibrils leading to the early establishment of an abnormal, scar-like, orientation. Moreover, the extracellular matrix of the fetal (and neonatal) dermis is less highly ordered, has a more rapid turnover and consists of less differentiated cells compared to the adult; these may enhance the scar-less healing pattern of the fetus. Interestingly, studies of wound healing in adult hair follicles have shown scar free healing of the damaged dermal papilla and the fibroblast-like cells of the papilla retain characteristics associated with embryonic cell lines, such as cell aggregation in tissue culture. It has been observed that wound healing is always faster and more fetal like on the oral mucosal side of the incision than on the skin side even in an adult. The adult oral mucosa possesses many embryonic characteristics and its structure is relatively simple by comparison with the complex, heavily keratinized, structure of adult epidermis. The epithelium of the adult oral mucosa is more fetal-like in its state of differentiation as reflected in the expression of cytokeratins. Human adult oral mucosal fibroblasts exhibit numerous embryonic characteristics and additionally, the oral mucosa is continuously bathed in fluid (saliva) rich in growth factors and antimicrobial agents, just like fetal skin and amniotic fluid.

However, the state of epithelial differentiation and potential epithelial-mesenchymal interactions during wound healing requires further investigation and a study of cytokeratins in fetal and adult wounds has recently been completed. Such a slow transition is indicative of the multifactorial differences between fetal and adult wound healing: rapidity of wound closure, degree of inflammatory response, maturity and competence of the immune system, degree of organization and differentiation of the wounded tissues, composition and organization of the extracellular matrix, presence or absence of an aqueous sterile environment (amniotic fluid) rich in growth (and other) factors, differences in tissue oxygenation, wound metabolism and pH, degree of tissue tension from function, presence or absence of acute inflammation, etc. Moreover, whether or not fetal wounds scar also depends upon the type and size of the wound (large, deep excisional wounds scar), the tissue wounded (internal tissues e.g. diaphragm, scar) and the species used—rabbit fetuses do not heal their wounds properly probably due to the large amounts of maternal immunoglobulin in rabbit amniotic fluid as a result of the size of the zona chorion during placentation. The differences between fetal and adult wound healing are multiple and subtle. There are studies to suggest a number of ways in which the healing adult wound might be experimentally manipulated to reduce scarring: enhance cell migration by addition of exogenous tenascin, develop a more open early wound scaffold to facilitate fibroblast migration and regeneration of the normal reticular collagen fibril orientation by addition of exogenous glycosaminoglycans and proteoglycans, clonally expand or transplant (e.g. from the oral mucosa) fibroblasts with embryonic characteristics at the wound site. It may be possible to manipulate adult wound healing to show a more fetal-like, scar-less, pattern by altering only one or two of the (major) components that are different between fetal and adult healing.

PIGMENT CHANGES IN SKIN FOLLOWING BURNS

The nature and extent of pigmentary changes in burned areas are not always predictable. The usual observation is that hyperpigmentation (Fig. 6.5) occurs after superficial burns whereas hypopigmentation/depigmentation are seen following deeper burns (Figs 6.6A to 6.7). Similarly, donor sites, where thin skin grafts are harvested, tend to hyperpigment later on. Whereas, if thick grafts are harvested, the donor area becomes hypopigmented. These changes are more noticeable in individuals with dark skin. There is enough literature on this subject as it has been extensively researched because the post burn pigmentary changes are a cause of considerable concern to the patients.

The pathophysiology for these pigmentary changes can be explained if the anatomy of normal skin is understood. The color of normal skin is determined by the number of melanocytes, the amount of melanin synthesized and its distribution and transfer to a keratinocyte. The melanocytes

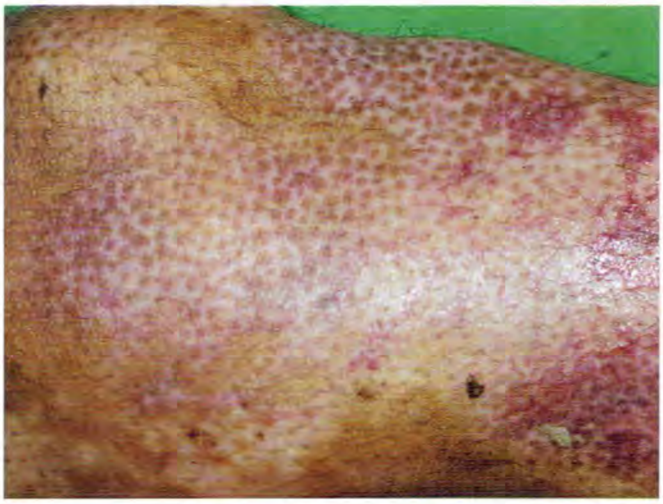
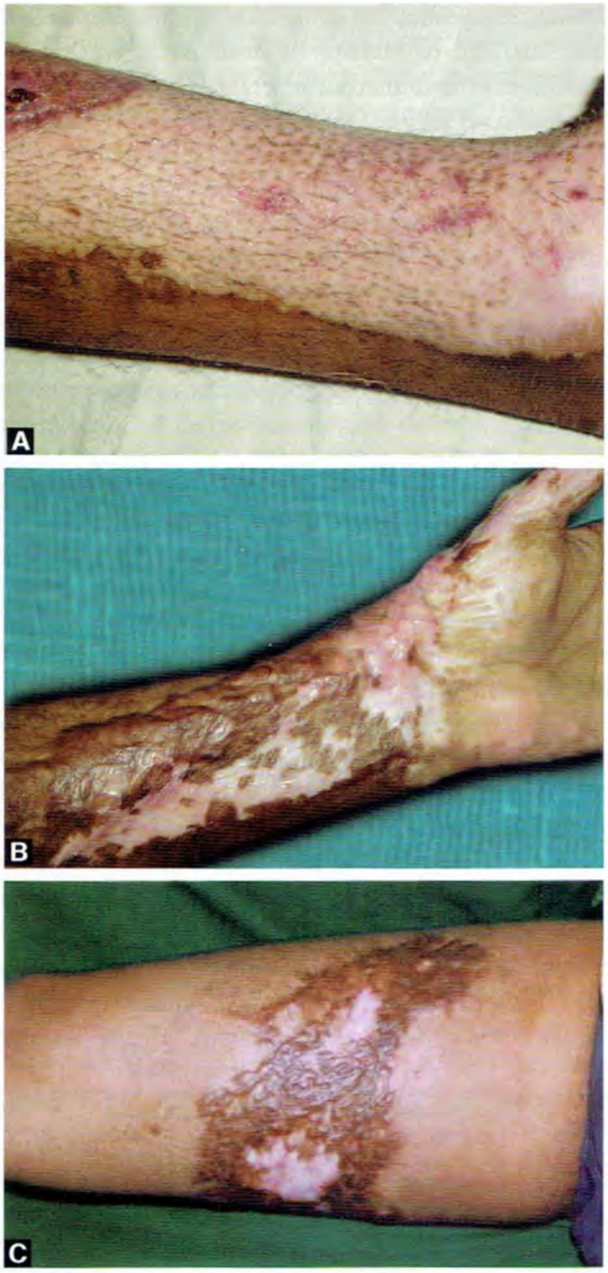


Fig. 6.5: Hyperpigmentation starting to appear in the scar



Figs 6.6A to C: (A) Hypopigmented area immediately after wound healed with reduced number of hair. (B and C) Hypertrophic scar showing hypopigmentation in the center where the wound was deeper and hyperpigmentation in the periphery where it was comparatively superficial

contain organelles, melanosomes, in which melanin is synthesized and then transferred to keratinocyte via cell to cell contact between melanocyte and keratinocyte. This is called the epidermal-melanin unit.

In case of superficial burns, the superficial layer of keratinocytes and melanocytes are destroyed but the melanocytes in the deeper layers of epidermis and dermis are still intact. They become overactive in response to the trauma of burns and stimulus of healing and probably synthesize more melanin. This leads to hyperpigmentation. This is also affected by the innate skin color, sun responsiveness of the individual's skin and amount of ultraviolet light exposure.

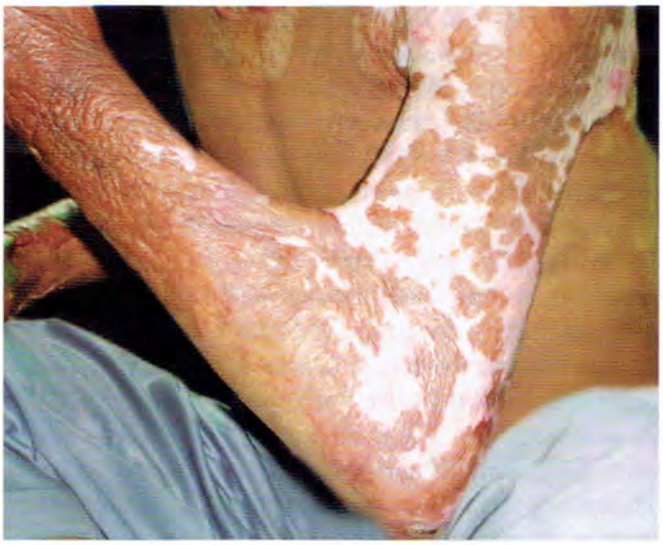


Fig. 6.7: Post burn contracture of elbow with hypopigmentaion in the scar

In case of deeper burns there are only few melanocytes left in the deeper layers of dermis. Therefore, there is a deficiency of melanin causing hypopigmentation or depigmentation in these areas after they heal. Only the residual melanocytes lining the intact hair follicles in the depth of the dermis migrate upwards to the basal layer of surface regenerating epidermis and give color to the healed skin which is deficient compared to surrounding healthy skin. Following skin grafting of raw areas, the re-innervation of the graft alters the melanocyte control and also increases the number of melanosomes in the epidermal-melanin unit leading to hyperpigmentation of the grafted areas.

HYPERTROPHIC SCARS AND KELOIDS

The word scar was derived from the Greek word *eschara*, meaning place of fire (fireplace). Scars are areas of fibrous tissue that replace normal skin or other tissues after injury. A scar results from the biological process of wound repair in the skin and other tissues of the body. Thus, scarring is a natural part of the healing process. With the exception of very superficial lesions, every wound (e.g. after accident, disease or surgery) results in some degree of scarring. Scar tissue is not identical to the tissue which it replaces and is usually of inferior functional quality. For example, the sweat glands and hair follicles do not grow back in the scarred skin (Fig. 6.8).

Examples of disfiguring scars include hypertrophied scars and keloids. In both keloid as well as hypertrophic scars, the wounds heal overzealously above the skin surface. The difference between a keloid and a hypertrophied scar is that whereas the former continues to enlarge beyond the original size and shape of the wound, the latter enlarges within the confines of the original wound (Figs 6.9 and 6.10). Although both can be red and raised, keloids continue to grow while hypertrophic scars tend to regress over time. Both can recur after surgical excision; however, keloids recur more commonly. Hypertrophic scars are far more common than keloids and may occur in persons of any age or at any site and tend to regress spontaneously. In general, they are more responsive to treatment. Keloids occur more frequently in black persons, being 5-15 times more common in black as compared to whites. They may occur in persons of any race with a proven tendency to keloid formation. This predilection for keloid formation in persons of certain races is not observed with hypertrophic scars. Keloids are more prevalent in persons aged 10-30 years while hypertrophic scars occur in individuals of any age. In general, the risk for either type of abnormal scar diminishes with age. Furthermore, the propensity for keloid formation can be familial, genetically transmitted as an autosomal dominant or recessive trait. For example, ear piercing custom prevalent in many cultures of the world, including India, can give rise to keloids (Fig. 6.11).

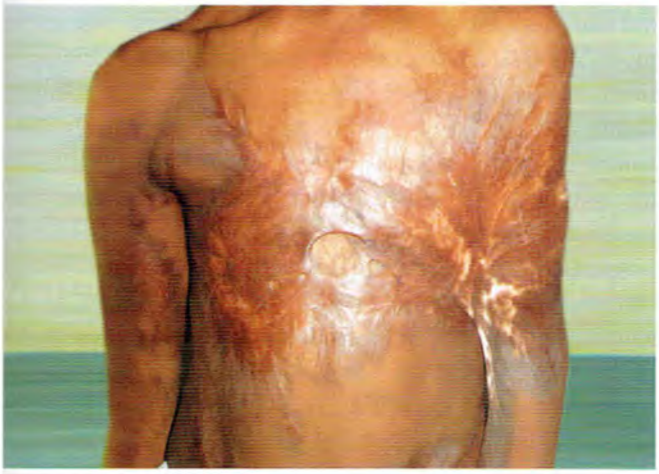


Fig. 6.8: Post burn scarring of chest with contracture of axilla. The scar is showing areas of both hypo- and hyperpigmentation

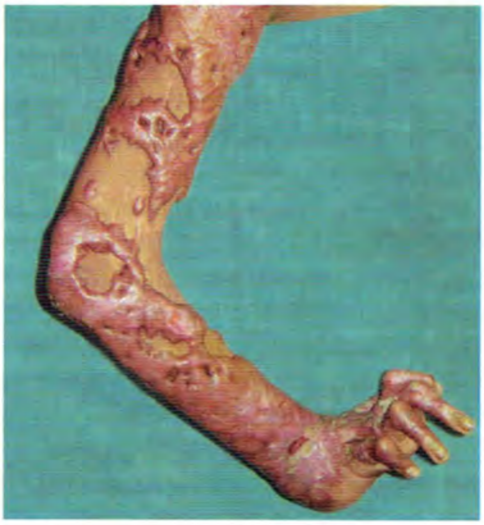


Fig. 6.9: Post burn keloid in arm with severe dorsal contracture of hand

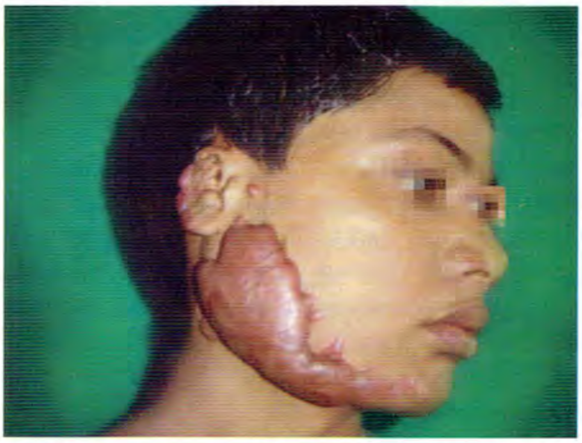


Fig. 6.10: Severe hypertrophic scarring over right side of face following scalds

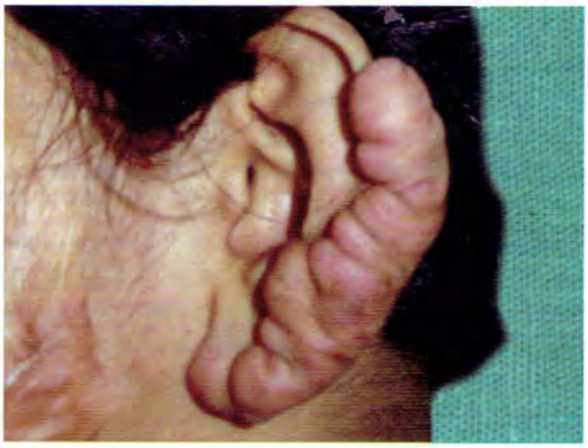


Fig. 6.11: Keloid over helical rim following burns

Etiology

Abnormal scar formation is unique to humans and as such, animal model research has not produced much significant information to further the understanding and treatment of keloids and hypertrophic scars. Despite numerous studies, no uniformly accepted theory or explanation indicates as to what initiates keloid or hypertrophic scar formation.

Several genetic and environmental causes have been implicated in the etiology of keloid and hypertrophic scars. In both, an excessive accumulation of collagen occurs either from increased collagen synthesis or decreased collagen degradation. Proposed causes for abnormal scar formation include foreign body reaction and bacterial infections. Almost all abnormal scars are associated with tattoos, burns, injections, bites, vaccinations, trauma, surgery or infection.

Skin tension is frequently implicated in hypertrophic scar formation. Abnormal scar healing commonly involves areas of high skin tension, such as the anterior chest, shoulders and upper back. Other factors implicated in the etiology of these scars include wound infection or anoxia, a prolonged inflammatory response and wound orientation different from the relaxed skin tension lines.

Immunologic alterations have been demonstrated in abnormal scars. Specifically, irregular immunoglobulin and complement levels, increased transforming growth factor-β and mast cells have been found in them. Additionally, decreased tumor necrosis factor and interleukin-1 levels have been found in these scars.

Pathophysiology

Although multiple factors are involved in their formation, studies indicate that keloid and hypertrophied scars result from increased collagen production and decreased collagen degradation. It was earlier demonstrated that levels of the collagen-related enzyme prolyl hydroxylase are elevated in keloid-affected skin compared to normal skin. Prolyl hydroxylase is required for the hydroxylation of proline during collagen synthesis suggesting that collagen overproduction occurs with keloids.

Collagen production is elevated in keloid biopsy samples and in cultured fibroblasts derived from keloids. Additional studies show that increased collagen production by cultured fibroblasts derived from keloids persists throughout their *in vitro* life span. No significant differences in DNA content or cellularity are found when keloid dermis is compared with normal dermis. This suggests that each fibroblast is producing more collagen rather than an increase occurring in the number of fibroblasts producing a normal amount of collagen. In keloid formation, excessive collagen production by fibroblasts may be due to the wound environment.

Abnormal wound healing in susceptible individuals can result in the formation of keloids that have an elevated content of extracellular matrix material compared to normal scars. Keloid-derived fibroblasts exhibit as much as a four-fold increase in the rate of fibronectin biosynthesis compared to fibroblasts from normal dermis and normal scars. Altered biosynthesis is due to an increase in the steady-state level of fibronectin mRNA. The level of fibronectin gene expression that is responsible for this increase has been identified. Thus, altered degradation of fibronectin mRNA is unlikely to contribute to overproduction of fibronectin in keloids.

The overproduction of fibronectin and type I collagen in keloids and hypertrophic scars implicates altered regulation of extracellular matrix components as an important aspect of these wound healing pathology.

Contrary to the increased collagen production theory, keloids and hypertrophic scars may also result from a decrease in collagen degradation. Levels of the collagenase inhibitor alpha-2 macroglobulin have been shown to be decreased in keloids.

The hypertrophic scars also have within them a large number of fibroblasts with contractile filaments in the cytoplasmic matrix and a deeply indented nucleus, indicating contraction of the cells. These fibroblasts with contractile properties are called myofibroblasts because they are structurally similar to smooth muscle cells. There is a marked increase in the number of these myofibroblasts in the granulation tissue and immature scars especially in the deep dermal portion of the hypertrophic scars. As these myofibroblasts contract, the stress on the adjoining collagen fibrils is released causing them to form compact collagen nodules which are seen in the depth of the dermis. The contracting myofibroblasts in the deeper dermis also cause the 'piling up 'of the scar as it is forced outwards. The increased number of myofibroblasts with their contractile properties also exerts sufficient force to cause severe distortion of facial structures and joint contractures as the new collagen fibers in the wound may fuse together in the position of comfort.

Clinical

On clinical examination, keloids and hypertrophic scars are raised above the skin level. Hypertrophic scars are

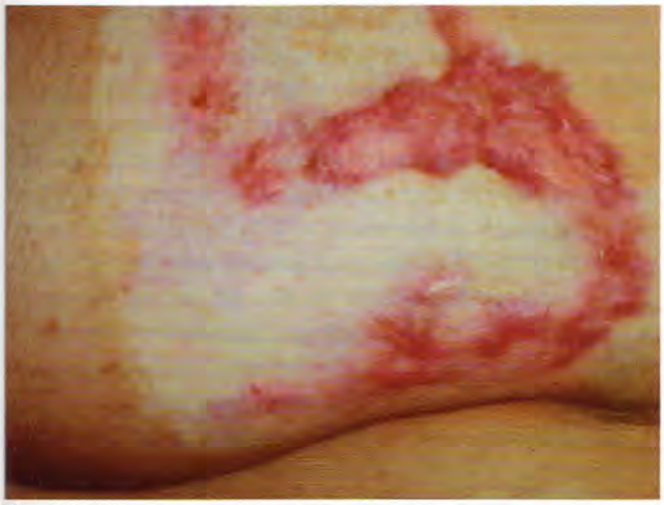


Fig. 6.12: Raised, red hypertrophic scar over chest following burns

self-limited; they hypertrophy within the confines of the wound (Fig. 6.12). Initially, hypertrophied scars can be raised, red, pruritic, and even painful; however, over time, they become pale and flat. Hypertrophied scars appear worst at 2 weeks to 2 months.

Medical Management of Hypertrophic Scars and Keloids

No single therapeutic modality is best for all hypertrophic scars and keloids. The location, size and depth of the lesion, the age of the patient and the past response to treatment determine the type of therapy used. Prevention is the key, but therapeutic treatment of hypertrophic scars and keloids includes occlusive dressings, compression therapy, intralesional corticosteroid injections, cryosurgery, excision, radiation therapy, laser therapy, interferon therapy, 5-fluorouracil (5-FU), doxorubicin, bleomycin, verapamil, retinoic acid, imiquimod 5% cream, tamoxifen, tacrolimus, botulinum toxin and other promising therapies such as transforming growth factor (TGF)–β3, and recombinant human (rh) interleukin (IL)–10 (rhIL-10), which are directed at decreasing collagen synthesis.

Prevention

Avoid performing nonessential cosmetic surgery in patients known to form keloids; however, the risk is lower among patients who have only earlobe lesions. Close all surgical wounds with minimal tension. Incisions should not cross joint spaces. Avoid making mid-chest incisions and ensure that incisions follow skin creases whenever possible.

Standard Treatments

These include occlusive dressings, compression therapy and intralesional corticosteroid injections.

Occlusive dressings include silicone gel sheets and dressings, non-silicone occlusive sheets and Cordran tape. These measures have been used with varied success. Anti-hypertrophic effect of silicone gel sheets appear to result from a combination of occlusion and pressure (especially when associated with pressure garments) and hydration. Studies to determine whether silicone compound itself affects the maturation of the scar have been inconclusive.

Compression therapy involves pressure, which has long been known to have thinning effects on skin. Reduction in the cohesiveness of collagen fibers in pressure-treated hypertrophic scars has been demonstrated by electron microscopy. Studies have shown that pressures of 15-40 mm Hg can be applied by custom made pressure garments and this reduces the reddish discoloration in the scar and also flattens and softens them after 6-12 months of continued application.

Corticosteroids, specifically intralesional corticosteroid injections, have been the mainstay of treatment. Corticosteroids reduce excessive scarring by reducing collagen synthesis, altering glucosaminoglycan synthesis and reducing production of inflammatory mediators and fibroblast proliferation during wound healing. The most commonly used corticosteroid is triamcinolone acetonide in concentrations of 10-40 mg/ml administered intralesionally with a 25- to 27-gauge needle at 4 to 6 weeks intervals.

Recent Innovations

Newer treatments for keloids and hypertrophic scars include intralesional interferon, 5-fluorouracil, doxorubicin, bleomycin, verapamil, retinoic acid, imiquimod 5% cream, tacrolimus, tamoxifen, botulinum toxin, TGF-β3, and rhIL-10.

Radiation Therapy

Using radiotherapy to treat keloids remains controversial. Although many studies have demonstrated efficacy and decreased recurrence rates, the safety of radiotherapy has been questioned. In one retrospective study of superficial X-ray therapy of 24 excised keloids, the author reported a recurrence rate of 53%. Use of iridium Ir^{192} interstitial irradiation after excisional surgery resulted in a 21%

recurrence rate after 1 year. Excisional surgery and preoperative hyaluronidase solution (150 U/ml sodium chloride) followed by external radiation (7.2-10.8 Gy) had a 0% recurrence rate. Adjunctive high dose rate brachytherapy (^{192}Ir) used after excision and closure resulted in a 12% recurrence rate after 26 months. When excisional surgery is followed by postoperative radiation therapy, the total fractionated dose should be a minimum of 12 Gy, according to a comparative study showing a higher recurrence rate for patients treated with total doses less than 12 Gy.

Other Potential Therapies

Additional potential therapeutic options for treating hypertrophic and keloidal scarring that have been shown *in vitro* to affect collagen synthesis include the use of proline-cis-hydroxyproline and azetidine carboxylic acid, tranilast (anti-allergic drug shown to decrease collagen and GAG synthesis) and pentoxifylline (inhibits DNA replication).

In addition, wounds treated with anti-TGFβ compounds healed with minimal scar tissue formation without affecting wound tensile strength. A possible candidate for affecting wounds via the neutralizing effect of TGF β is the proteoglycan termed decorin.

Surgical Management of Hypertrophic Scars and Keloids

Cryotherapy

Cryosurgical media (e.g., liquid nitrogen) affects the microvasculature and causes cell damage via intracellular crystals, leading to tissue anoxia. Generally, 1, 2, or 3 freeze-thaw cycles lasting 10-30 seconds each are used for the desired effect. Treatment may need to be repeated every 20-30 days. Care is taken to administer liquid nitrogen in short application periods because of the possibility of reversible hypopigmentation. Cryotherapy can cause pain and permanent depigmentation in selected patients. As a single modality, cryosurgery led to total resolution with no recurrences in 51-74% of patients after 30 months of follow-up observation.

Excision

For excision of small hypertrophic scars, closure with minimal tension, paralleling the relaxed skin tension lines is carefully planned.

Use buried sutures when necessary for layered closure and to reduce tension. Whenever feasible, apply pressure dressings and garments during the immediate postoperati
period to wounds of patients in whom hypertrophic sc
and keloid formation occur. Decreased recurrence rates ha
been reported with excision in combination with oth
postoperative modalities, such as radiotherapy, inject
interferon, or corticosteroid therapy. Excisional surgery alo
has been shown to yield a 45-100% recurrence rate a
should very rarely be used as a solitary modality, althou
excision in combination with adjunct measures can
curative. Most studies in which excisional surgery w
combined with injected steroids indicated less than 50
recurrence.

Shaving and over grafting has been used for seve
hypertrophic scarring which causes functional restricti
also. This involves shaving the hypertrophic scar by usi
a manual skin grafting knife till the entire fibrous tissue
removed and healthy reticular dermis is reached, over whi
a thin skin graft is applied. This usually helps in release
minor contractures as well.

Laser Therapy

Ablation of keloids and hypertrophic scars using a carb
dioxide laser (10,600 nm) can cut and cauterize the lesi
creating a dry surgical environment with minimal tiss
trauma. When used as a single modality, the carbon dioxi
laser was associated with recurrence rates of 39-92% a
when it was combined with postoperative steroids injecti
it was associated with recurrence rates of 25-74%.

The argon laser (488 nm), similar to the carbon dioxi
laser, can induce collagen shrinkage via generation
excessive localized heat. The argon laser has demonstra
recurrence rates of 45-93%. The pulsed dye laser (585 n
provides photothermolysis, resulting in microvascu
thrombosis. Beginning in the 1980s, authors noted that sc
became less erythematous, more pliable, and l
hypertrophic after treatment with the 585 nm pulsed
laser. The findings were later confirmed using object
measurements of erythema by reflectance spectrome
readings, scar height, and pliability measurements. Beca
of its efficacy, safety, and relatively low cost, the pul
dye laser remains the laser treatment of choice
hypertrophic scars.

CONTRACTURES

A contracture (Figs 6.13 and 6.14) is a tightening of
that may affect the underlying muscles and tendons limi
the joint mobility. Contractures develop when normal ela

connective tissues are replaced with inelastic fibrous tissue. This makes the tissues resistant to stretching and prevents normal movements of the affected area. Physical therapeutic measures can aid in controlling burn scar contractures. If these treatments do not control the effects of contractures, surgical intervention is required. After release of contracture, a skin graft or a flap cover is provided. The management of this post burn complication is discussed elsewhere in detail in this volume. Conservatively managed deep dermal burn wounds and delayed skin grafting of full thickness burns commonly end up in various kinds of scar deformities and contractures. This significantly restricts physical and social rehabilitation of a burn victim.

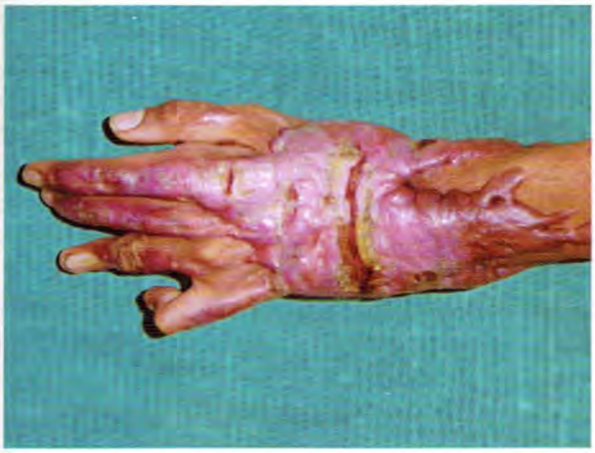


Fig. 6.13: Post burn hypertrophic scar over dorsum of hand and distal forearm leading to contracture at wrist and fingers with deformity of fingers

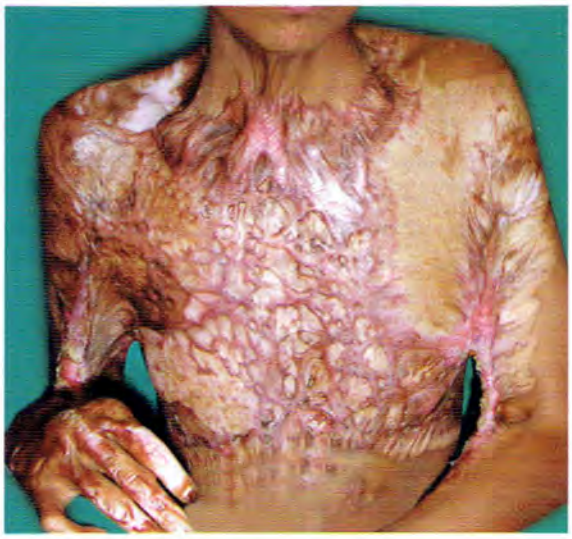


Fig. 6.14: Hypertrophic scarring over chest causing contracture of neck and both axilla

Skin scar contractures due to destruction of skin are very frequent and when present over joints lead to contractures of joint. Secondary contractures cause shortening of musculo-tendinous units and neurovascular bundles and of tissues around a joint leading to decreased range of movement. Primary arthro-osseous contractures result from direct deep burns in a joint, leading to severe and irreversible processes. Contractures can occur at any joint of the body. This joint dysfunction can also be a result of immobilization from burn injury. A contracture is different from spasticity, but they are often related. Spasticity is an abnormal increase in muscle tone. This can worsen the development of contractures.

SUMMARY

Skin is the largest organ in the human body forming its outer protective covering like a shield. When it is damaged in the case of a burn patient, it alters even the internal milieu. Therefore, it is important to restore its structure and function as soon as possible by conservative and surgical means. In order to achieve this goal, it is essential to understand the various biological processes which tend to heal the skin wounds and how they can be hastened. The wound healing response is by and large predictable with some degree of variability. Sometimes, the healing processes go haywire as is evident from development of hypertrophic scars and keloids. Newer modalities are being explored to maximize the rate of wound healing and minimizing the scar.

Webster states "The great ignominy of the plastic surgeon is his inability to remove a scar without leaving another one. The best we can do is to occasionally improve on another surgeon's scar while indeed he may be improving on several of ours."

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Chapter 7

Immunological Responses to Burn Injury

Shankar Sriniwasan

Infection continues to be the leading cause of death in burn patients. Increased susceptibility to infection arises from the large open wounds, increased metabolic requirements, decreased nutritional intake, loss of proteins into the burned areas and suppression of host immune response.

Alteration of host defense mechanisms in thermal injury is a complex interaction of nutrition, hypermetabolism and immunological alteration leading to a complex tango of events which, to date, is an unresolved problem.

Nonspecific Immunity: Skin is an important defense mechanism against microbiological invasion. Any breach in this barrier is a harbinger for infection and its attendant complications.

Any organism which gains entry must first contend with local conditions at the point of entry. Vasoactive amines like histamine and serotonin, kinin polypeptides and various other mediators initiate a local inflammatory response. They cause constriction of venular sphincters and dilatation of capillaries, resulting in increased capillary permeability. Thus, fluid and fibrinogen leave the dilated vessels and create a fibrin network which localizes bacteria. Leucotaxis is also aided by the kinin system.

Serum has two classes of protein:

1. Beta lysine which is lethal to gram-positive organisms.
2. A group of proteins called complement which acts against gram-negative organisms.

If local mechanisms do not stem the infection, the microorganisms are transported to the lymph nodes by the lymphatics where macrophages phagocytose the organisms. The specific immune responses are now called into play at this location by the lymphocytes.

Specific Immunity: It can be discussed under two subdivisions: cell mediated and humoral.

The lymphocyte is the key cell in the initiation of specific immune response. It is the only cell capable of recognizing an antigen as foreign and initiating specific mechanisms for its disposal.

Lymphocytes form two response systems:

1. T cell, associated with cell mediated immunity.
2. B cell, associated with humoral or antibody mediated immunity.

Both T and B lymphocytes arise from a common bone marrow stem cell. On maturity, the T and B lymphocytes seed the peripheral lymphoid organs. It must be emphasized here that there is a complex interaction between these systems and that any simplified description is only to aid in the understanding of this complex process.

Cell mediated immunity (CMI): Briefly, the cell mediated immunity when stimulated, causes the T-lymphocytes to undergo blast transformation and proliferate. T cell cooperation is needed for B cell responses to occur. T cells also secrete a variety of lymphokines which regulate immune responses. Lymphokines also activate macrophages to increase microbicidal activity.

Altered CMI following burns has been well documented and researched. This is evidenced by prolonged survival of allografts in extensively burnt patients. There is a decrease in total counts and increase in suppressor T cells (lymphocytes also have a depressed responsiveness owing to generation of a subpopulation of T cells called suppressor T cells).

Humoral immunity: The hallmark of humoral immunity is the production of antibodies by the B lymphocytes. Antibodies which have significant activity against microorganisms are IgG, IgM and IgA.

All immunoglobulins are decreased in quantity during the first week post burn and return to normal levels during the second week after the injury. The decrease in IgG levels is more than IgM or IgA. The initial decrease is followed by higher than normal concentrations owing to antigenization which burn patients are subjected to during their clinical course. Serum IgM level is elevated owing to widespread fungal infection in these patients.

Complement system: It is a complex integrated network of serum proteins that bridges both specific and non-specific immunity. On activation, they mediate inflammation and tissue damage and facilitate phagocytosis. Edema formation in injured tissue is complement dependant. Complement components C3a and C5a facilitate serotonin and histamine release which cause vasodilatation and thus edema.

A co-ordinated immuno-inflammatory response requires the regulation of many cell types achieved by the actions of soluble protein (cytokine) and lipid or peptide (autacoid) mediators released by macrophages, neutrophils, lymphocytes, platelets and endothelium. Some cytokines like tumor necrosis factor alpha, interleukin-1 (IL-1) and IL-8 have pro inflammatory actions while IL-4, IL-10 and IL-13 have counter regulatory activities. Some cytokines (e.g., IL-1) are released early while IL-6 and IL-8 appear later in the pathological continuum.

Cytokines and autacoids regulate the amplitude and duration of the immune response. They have multiple effects on cell differentiations and there is considerable overlap in their actions. To attempt to control and localize an inflammatory process, both pro- and anti-inflammatory mediators are released simultaneously. A balance between these two competing mechanisms is critical if homeostasis is to be maintained. Their often compartmentalized production results in serum concentrations which do not reflect accurately their local physiological levels. Quite aptly, Cavaillon et al, have described serum cytokine concentrations as being tip of the iceberg.

IMMUNOMODULATORY THERAPIES

The treatment of severe sepsis and septic shock has centered around the eradication of established infection with surgery and antibiotics along with intensive organ support. With the realization that the pathological effects of sepsis can result from the direct actions of micro-organisms and their toxins and indirectly from the host response, interest has been sparked off in newer immuno modulatory therapies. These therapies have attempted to attenuate the actions of bacterial toxins, limit host inflammatory response or to reduce tissue damage resulting from established inflammatory processes. Anticytokine therapies have attempted to inhibit pro-inflammatory or enhance anti-inflammatory responses. In all this, a delicate balance needs to be achieved between inhibition of an excessive response and ablation of essential host defenses. Derangement of either can lead to systemic sepsis and death.

Endotoxin

Endotoxin or lipopolysaccharide (LPS) is found within the external membrane of the cell wall of gram-negative bacteria and is released from growing and damaged microorganisms. LPS binds to many serum carrier molecules, the most important of which is lipopolysaccharide binding protein (LBP). The LPS-LBP complex interacts with macrophages via the CD14 cell-surface receptor. LPS is a potent stimulator of cytokine production. Administration of endotoxin to healthy human volunteers produced clinical features of sepsis.

Anti-endotoxin therapies: In early studies, polyclonal human antisera with high levels of anti-LPS antibodies were shown to reduce the mortality arising from gram-negative sepsis. With technological advances came anti-LPS monoclonal antibodies (e.g., HA-1A and E5). Despite encouraging animal study reports, large, random, controllec trials in humans have been disappointing.

Tumor Necrosis Factor Alpha (TNFα)

It is a polypeptide produced predominantly by the cells o the reticuloendothelial system. It has a short half life and pivotal role in the expression of sepsis. It acts directly o cells and stimulates the release of other mediators o inflammation.

Anti-TNF alpha therapies: Anti-TNF alpha monoclon antibodies are some of the most promising therapeuti modalities available. Unfortunately, this did not bear out i some large scale studies.

The Interleukins (IL)

The interleukins are a class of cytokines produced by mar cell types. IL-1 and IL-8 have pro-inflammatory actic while IL-4 and IL-10 have anti-inflammatory actions. Il 6 is regarded as the best available serum marker of tl severity of inflammation. IL-1 is a key mediator in tl immuno-inflammatory cascade, interacting closely with other cytokines, particularly TNF alpha.

Anti-IL-1 therapies: IL-1Ra (IL-1 receptor antagonist) is the most extensively investigated therapy. Unfortunately, as with other therapies, it has not been found to be effective clinically.

Other Potential Immunomodulatory Therapies

The autacoids are important mediators in the immuno-inflammatory cascade and include arachidonic acid derivatives-prostacyclin, thromboxane A2, the prostaglandins (particularly PGE2) and the leukotrienes.

1. Cyclooxygenase (COX) is an enzyme central to the production of these derivatives and COX inhibitors appear to have some benefits in septic patients. Ibuprofen was tried but conferred no survival advantage.
2. Selective TXA2 inhibition may be preferable to non-selective COX inhibition as prostacyclin synthesis would be maintained. The azole anti-fungal agent ketoconazole is a selective thromboxane synthetase inhibitor and a reduction in mortality has been observed in a small placebo controlled trial.
3. Platelet activating factor (PAF) is a pro-inflammatory mediator produced by the endothelium, neutrophils and platelets. Treatment with the naturally occurring PAF antagonist, BN 52021 has produced encouraging results.
4. Bradykinin is a potent pro-inflammatory mediator that is a potent stimulator of other autacoids and nitric oxide. The bradykinin antagonist, CP 0127 has been tried and shown to be useful in a small population of patients with gram-negative infection.
5. Nitric oxide synthetase inhibition.
6. Fungal glycans: They increase macrophage microbicidal activity and induce monokine release, e.g. glucan.
7. Thymic extracts, e.g. thymopentin(TP-5)is licensed for use and works by its ability to activate T cell population. It decreases prostaglandin E2 production. PGE2 is an immunosuppressant and inhibits T cell activation.
8. Granulocyte colony stimulating factor has been shown to have benefit.
9. Decreasing bacterial translocation following burns can be achieved by giving prostaglandin analogues, e.g. misoprostol, enisoprost.
10. Interferon gamma
11. Lipopolysaccharide antagonist: polymyxin B. It absorbs LPS and prevents endotoxemia.
12. Free radical scavengers like superoxide dismutase improve survival in animal models.

The complexities of the immunoinflammatory cascade are such that pharmacological intervention has proved difficult and cumbersome, not to mention prohibitively expensive and beyond the reach of the common man. Despite encouraging results from animal studies, no therapy has shown to produce an unequivocal reduction in mortality in clinical sepsis trials. It is unlikely that any single agent will do the trick and the early use of immunostimulants followed by the use of immunosuppressants later may prove to be the most useful approach. It is nevertheless certain that the benefits are likely to be small and unlikely to be realized in the absence of optimal resuscitation, surgery, antibiotics and intensive care therapy.

NUTRITIONAL IMMUNOMODULATION

Three categories of products have shown promising results in chronic burn patient:

1. Long chain fatty acids
2. Arginine, glutamine and branched chain amino acids.
3. Nucleotides.

Amongst all above, glutamine is the best studied. It is the most common amino acid in the body and is used by rapidly dividing cells. Its administration supports the gut barrier and minimizes gut translocation of bacteria and thus the catastrophic complications arising from breakdown of the gut barrier is mitigated.

IMMUNOTHERAPY

In 1964, Kefalides noted a decrease in serum levels of gamma globulin in burned children. The levels returned to normal in 7 to 9 days.

The general consensus was that there was a marked drop in IgG levels immediately post burn which gradually returned to normal approximately two weeks after the insult.

The severity of suppression of IgG was related to burn size and the degree of depletion of IgG could be correlated to mortality from sepsis.

IgG concentrations under 300 mg% within a day or two of admission carry a very poor prognosis.

As an adequate concentration of IgG is critical to the outcome, it is reasonable to:

1. Stimulate production by vaccination.
2. Increase serum concentration by passive immunization.
3. Combination of the above.

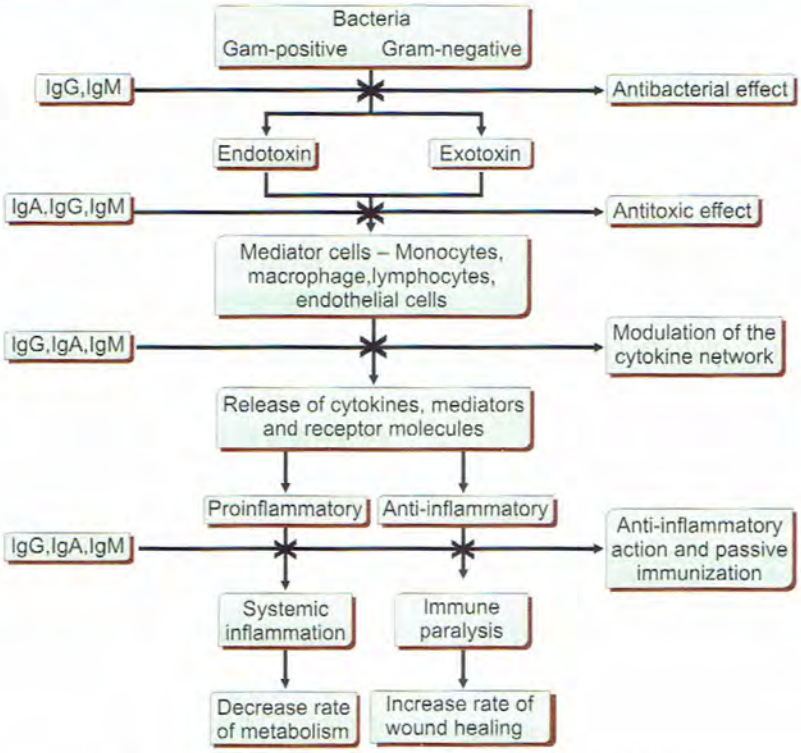
Bacteria
Gam-positive
Gram-negative
IgG,IgM
Antibacterial effect
Endotoxin
Exotoxin
IgA,IgG,IgM
Antitoxic effect
Mediator cells – Monocytes, macrophage,lymphocytes, endothelial cells
IgG,IgA,IgM
Modulation of the cytokine network
Release of cytokines, mediators and receptor molecules
Proinflammatory
Anti-inflammatory
IgG,IgA,IgM
Anti-inflammatory action and passive immunization
Systemic inflammation
Immune paralysis
Decrease rate of metabolism
Increase rate of wound healing

Fig. 7.1: Rationale for immunotherapy with IV immunoglobulins

Early attempts centered on the development of vaccines against *Pseudomonas aeruginosa*. This is not in widespread use today because the incidence of life threatening infections due to *Pseudomonas* infection have fallen and for the vaccine to be effective,it is necessary to vaccinate the patient several days before infection becomes a threat to get sufficient antibody levels in the serum. This luxury of time is often not available.

Passive immunization: It holds a lot of promise. It is attractive because the product is immediately available and effective at the time of administration. Cutter® Iv Ig was first tried in the US and was successful. It was 90% IgG fractionated from a pool of donors. It had excellent antibody titres against all types of *Pseudomonas aeruginosa* along with useful titres against streptococci, *E. coli*, *Staph. aureus* and some viruses too.

Pentaglobin®, i.e. IgM enriched intravenous Immunoglobulins have an anti-bacterial, anti-toxic, anti-inflammatory and immune modulating activity.

Pentaglobin embodies natural principles of physiological defense mechanisms and presents a rational basis for the therapy of sepsis. Hence, it reduces rate of metabolism and energy requirement.

CONCLUSION

Active vaccination is not practical because of changi spectrum of pathogenic microorganisms in burn patient

Passive immunization holds a lot of promise. A high tit polyvalent immunoglobulin product that offers protecti against gram-negative organisms (other than *Pseudomona* is desirable.

A combination of topical and systemic antibiotics, bett nutrition and immunomodulatory methods will play a decisi role in the management of burn patients in the foreseeab future.

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Chapter

8

The Immunobiology of Skin Transplant Rejection and Acceptance

Rakesh Kain

INTRODUCTION

Attempts at skin transplantation have been recorded over the centuries, often in graphic detail. The earliest reports of transplantation in humans included several miracles. One of best-known examples is the thirteenth century story of Cosmos and Damian. They were Christian Arab saints who were martyred around 300AD and were reputed to have successfully replaced the diseased leg of sexton with that from a moor who had died several days earlier. The practice originated in India and was modified to great effect in Europe.[1]

The immune system identifies and attempts to destroy all cells and tissue recognized as foreign. Only the tissues from genetically identical donors are exempt as they do not express foreign antigen that can initiate a response. Even the smallest disparity, a single amino acid in a major histocompatibility (MHC) molecule, can bring about the skin transplant rejection in mouse. The transplantation barrier can be defined in terms of genetic difference(s) between the donor and the recipient and this is discussed later. In general terms, there are both antigen specific and non-specific responses including antibody mediated, T-cell - mediated and inflammatory reactions.

To reduce transplant rejection, one either avoids transplantation into a sensitized recipient or minimizes the antigenic disparity between the donor and the recipient or else, treats the patient with various immunosuppressive agents. Knowledge of the immune response to transplanted tissue provides a rationale for the use of currently available agents and may help in the design of new products/ protocols for future use.

THE SCIENTIFIC BASIS OF TRANSPLANTATION

The modern era of transplantation began in 1950s. However, the preceding decade was a defining period for transplantation because it was during this period when Peter Medawar and colleagues showed that the loss of a tissue allograft was brought about by the immune response it provoked in the recipient and not as a result of some kind of non-specific inflammatory reaction(s).[2]

With the aid of grant from the Medical Research Council, Medawar travelled to Glasgow to study skin grafting in the burns unit at Glasgow Royal Infirmary. There, he worked with Tom Gibson, a gifted plastic surgeon, who was interested in the study of skin graft rejection. Shortly after Medawar's arrival, a young lady was admitted to hospital with severe burns after falling on to an open gas fire. Gibson grafted the women's wounds with a series of small pinch skin grafts taken from her brother and Medawar proceeded to study the fate of the skin grafts by taking their biopsies for histological examinations.[3] As expected, the grafts were destroyed after some days and when a second set of graft from the same donor was applied 2 weeks later, they were destroyed even more quickly. This so called "second set" phenomenon was taken as clear evidence that the rejection response was due to actively acquired immunity and not to a non-specific inflammatory reaction. Medawar and Gibson published these findings in the journal of anatomy (1944).[4] They concluded that an, as yet, unidentified antibody is responsible for rejection.

Regardless of whether antibody or cellular immunity was responsible, there was now widespread acceptance that immunological mechanisms were the cause of graft rejection. Immunological rejection was viewed as an inevitable consequence of organ transplantation and unfortunately no effective way of preventing rejection was known. Attempts to surpass graft rejection had met with, at best, minimal success.

Behavior of Transplanted Tissue

One important point to bear in mind is that the graft responds to its environment in the recipient. The organ is not inert. It changes its expression of cell surface and produces a range of cytokines and other factors. It does so in response to physiological stress and immune attack. These normal tissue responses of graft influence the outcome of transplantation, by increasing graft antigenicity and immunogenicity.

TRANSPLANTATION AND GENETIC BARRIERS

The degree of antigenic disparity between the donor and recipient is important and can be defined in terms of genetic differences between individuals. Donor tissue carries a large number of different molecules that may be recognized as foreign by the immune system of the recipient. These include the blood group antigens to which there are preformed antibodies. Then there are major histocompatibility (MHC) molecules that may be recognized directly by the receptors on recipient T cells. In addition, there is all the protein of the tissue that can be degraded to peptides and can activate cellular responses in the recipient (Table 8.1).

THE IMMUNOGENIC ELEMENTS

Donor tissue carries a large number of different immunogenic molecules which may be the functional cells of the graft. These molecules are recognized as a foreign substance by the immune system of the recipient. Together they carry out one or more different functions. These cells usually express only low level of immunogenic response. These include the blood group antigen to which there are preformed antibodies. Other is MHC molecule that may be recognized directly by the receptors on recipient T cells. The endothelial cells form the lining of all the vasculature present in whole of the body. The endothelium is usually quiescent but can be activated in a variety of ways by cytokines and by cell-cell contact. The endothelium contains a number of adhesion molecules called selectin and integrins. IFN-γ a cytokine and TNF-α alter endothelial cell expression of adhesion molecule and increase the expression of both MHC 1 and MHC 2 antigens on endothelial cells. Hence endothelium, especially after activation, is highly antigenic.[5]

THE MECHANISM OF GRAFT REJECTION

The two immune systems operate together. One is an innate immune system which combines cells of monocyte and granulocyte origins, macrophages, neutrophils and eosinophils, along with complement components, to provide a simple phagocytic and inflammatory defense against bacteria and other organisms. Another immune system is "adaptive immune" system which involves the activation of two classes of antigen-specific lymphocyte. The B cells are derived directly from bone marrow and form antibodies against antigens.[6] The T cells originate in the bone marrow but pass through the thymus and go on to recognize peptide fragments of antigen present on the surface of strongly MHC positive antigen presenting cells. Once activated, the B and T cells can be more readily activated so that second or subsequent exposure to the same antigen elicits a bigger and more rapid response.[1]

The immune system operates by importing a vast armamentarium to the site of inflammation. The number of possible reactions and combinations of immunological components that come into play to destroy a transplant has led to some difficulty in understanding the process fully.

The Recipient in the Graft

During rejection process, cells have to migrate from the blood stream in to the transplanted tissue. Endothelium

Table 8.1: Genetic relation affecting outcome of transplant

Transplant	*Donor –patient relationship*	*Outcome of transplant*
Autograft	Same individual	No foreign antigen, therefore, no rejection
Isograft	Genetically identical donor and recipient (monozygotic twin)	No foreign antigen, therefore, no rejection
Allograft	Genetically different individuals of same species (the usual clinical situation)	Recognition mainly of mismatched donor MHC antigens. Vigorous rejection without MHC matching and immunosuppression.
Xenograft	Different species (e.g., pig to human)	In some species combination there are preformed antibodies,leading to hyperacute rejection, the greater the phylogenetic gap the more vigorous the cellular rejection response.

expresses the adhesion molecules called E-selectin, P-selectin, etc. which appear on surface within seconds of stimulation with thrombin, histamine and other agonists. This causes leukocytes to slow down and roll along the endothelial cell surface followed by activation of integrin adhesion molecules. This and other mechanisms lead to firm adhesion of leukocyte to endothelial cells and their flattening against the vessel wall.

SKIN ALLOGRAFT REJECTION AND ACCEPTANCE

Skin is a strongly antigenic organ due to 'skin specific antigen', an antigen presented by the skin graft after it is transplanted. Hence, it is subject to rejection.

The obvious alternative to split skin autograft and the problem of limited availability of donor sites is skin allograft. Though cultured keratinocytes can be used as substitutes for skin allografts, the facility and infrastructure for skin culture is not available in many of the developing countries. For such places, the only skin substitute that can be used in extensive burns where enough donor areas are not available, is allograft, taken from the burned patients' close relatives, particularly mother, father or siblings. However, all allografts from random donors start getting rejected in about 2 week's time. Even those obtained from parents start getting rejected in about 3 weeks if patient is not severely immunocompromised. Rejection of full thickness skin allograft by immunocompetent individuals is such a consistent and reproducible phenomenon that it has been the central model for the study of immunologically mediated tissue destruction *in vivo* since the work done by Medawar (Gibson and Medawar[7], 1943). The only documented permanent coverage with skin allograft in humans has been in identical siblings (Converse and Duchet[8], 1947; Stranc[9], 1966; Caruso et al[10], 1996).

Skin graft revascularization is the basis for the beginning of graft acceptance or rejection or both. The vascularization of graft begins immediately after a graft is applied to the host bed, be it an autograft or an allograft. By 6 hours, only the plasmatic imbibition nourishes the graft, although the host endothelium starts to proliferate. By 9 hours, inflammatory cells invade the graft. By 12 hours, the host endothelium penetrates the fibrin interface between graft and host and small communications start forming between graft and host vessels. This is called inosculation. By 24 hours, the host vessels grow into the graft and vascular communications are well established by 48 hours. By 4 days the arterial end of the capillaries are open and slow arterial flow from the host to the graft begins. The circulatory outflow from the graft to the host (from the venous end of capillaries) starts around the 7th day and this brings the donors antigens into the host circulation in case of allograft. The host then produces antibodies against the graft. By 10th day, this starts the rejection phenomenon and the process of rejection becomes clinically evident by the 14th day in the form of maceration of the graft. In fact, there are 2 types of reactions which take place following allograft transplant. The first is cellular reaction which is local and the evidence for the failure of homograft is found within the graft themselves or their immediate surroundings. The second reaction is humoral reaction which is systemic in nature and follows a general pattern of antigen –antibody reaction. Corollary to this is that if homograft is applied and removed before venous return is established, i.e. 7th day and then reapplied to the same or different area, it can survive for another 14 days till it is rejected again.

White graft rejection: when homograft is applied to a burn patient for a second time from the same donor, it has been found that it stays white and does not turn pink as no circulation is established in this graft and therefore, it is not taken up as in normal circumstances. The explanation for this is that once a homograft is applied to the host for the first time, antibodies are formed against the homograft and they stay in the host circulation from that time onwards. On second application of the same homograft, it is immediately rejected by the host and not taken up at all. In other words, the graft remains white. This is called "White rejection". This is also known as 'Second set rejection'.

In case of major burns, the immunocompromised state of the patient can delay rejection of allografts for several weeks.

However, allograft skin is used as a biological dressing in the management of severely burned patient. Thus widely meshed (>1:6) split skin autograft is interleaved with allograft either by overlaying (Alexander et al[11],1981; Phipps and Clarke[12], 1991) or insertion of allograft in holes cut in autograft sheets(Yang et al[13], 1979). Except for one isolated case report (Takiuchi et al[14], 1982) allografts do not survive in the long term, the rejection always occurring before 3 months and often within the first 3 weeks of transplantation. Attempts to modify this outcome with general immunosuppression have been made (Burke et al[15,16], 1974, 1976) but encountered a serious problem with sepsis as a result of immunodeficiency.[17]

Experimental studies in animal model showed that skin allograft survival was prolonged by cyclosporine (Borel and Meszaros[18] 1976; Black et al[19] 1988). However, the actual fate of the allograft in many of these studies is not clear. In 1989, Frame et al[20] treated patients with cyclosporin after allografting .The allograft survived during drug treatment but were rejected in two cases after cessation of therapy.[1]

TYPES OF REJECTION

According to time interval and host response against graft, the rejection can be classified into four categories:

1. **Hyperacute rejection (minutes to hours):** Occurs within minutes to hours of transplantation. Preformed anti-donor antibodies cause complement fixation and thrombosis in the vasculature of graft.
2. **Accelerated acute rejection (1-4 days):** Refers to the situation where the T cells of the recipient have been immunized against antigens of the donor by prior exposure to foreign cells and tissues (presensitized T cells also known as "second set" rejection).
3. **Acute rejection (days to weeks):** Acute rejection occurs within days to months after transplantation. In fact the period of greatest risk of acute rejection extends to about 3 months after grafting. The principle immune response is activation of T- lymphocytes. These cells are of two subtypes—CD-4 positive cells or helper cells (Th) and CD-8 positive cells or cytotoxic cells (Tc). The Cd-4 cells may have cytotoxic activity and CD-8 cell makes a range of cytokines.
4. **Chronic rejection:** Chronic rejection generally occurs months to years after transplantation. Defining chronic rejection is difficult against a background of other processes that are going on at the same time. Immunological mechanisms of chronic graft dysfunction include the binding of antidonor antibodies and sub-acute cell mediated damage. The sites of the damage are the vasculature and the parenchyma. On-going immunological mechanisms of graft vasculopathy include hypertension, hyperlipidemia, and cytomegalovirus infection, etc. probably mediated by release of various growth factors within the transplant.[5]

GRAFT VERSUS HOST DISEASE (GVHD)

Transplantation between genetically non-identical individuals is, at present constrained by two major immunological responses. First is graft rejection and second is graft vers host disease (GVHD).[21]

In a burn patient whose immunity is very low, e.g. if tl patient is in sepsis, even if homografts are applied repeated to the host's raw areas, no antibodies are formed agair the graft by the host because of low immunologic response. Therefore, the graft is not rejected.[22] Instea gradually the homografts antigens start becoming strong and they mount an immune response against the host. Tl host's antigens become foreign to the homograft and tl homograft starts forming antibodies which destroy the hc cells. This is called 'Graft Versus Host disease' (GVHD) 'Runting syndrome'. Patient may manifest with anemi weight loss, fever, edema, ulceration, loss of hair ar spleenomegaly.[23]

Acute GVHD is characterized by a distinctive syndron comprising dermatitis, hepatitis and enteritis. The severi of disease is defined by a grading scheme from 0 to 5.

0 and 1are mild disease
2 is moderate
3 and 4 are severe and/or life threatening
5 is life threatening

Even with optimal immunosuppression, patient shov significant morbidity and mortality. In long term after sk transplantation, after 100 days, a different form of GVH may emerge even though initially it may have bee considered as successful graft 'take'. These are considere as autoimmune phenomenon with relative immunodeficien (Herman Waldman[22], 1995). Experimental work in anim models shows that both forms of GVHD can be caused b both CD4 and CD8 cells.

Treatment for Graft-versus-Host Disease (GVHD):

1. UVA-1 Phototherapy: Clinical responses of sclerotic sk to UV-A1 phototherapy were modest cause of UV-A1 induced skin darkening, which is photo-protective ar attenuates antifibrotic responses.
2. Low dose Methotrexate: Methotrexate (MTX) appea to be a well-tolerated, inexpensive and possibly steroi sparing agent that is worthy of further evaluation GVHD.
3. Extracorporeal Photo-chemotherapy (ECP), considere as an effective treatment for patients with erythroderm cutaneous T-cell lymphoma, has recently been use successfully in the treatment of GVHD and prospecti trials for treatment of chronic GVHD.
4. Anti-inflammatory proteins in combination with steroic

CULTURED KERATINOCYTES ALLOGRAFT

Autogenous cultured keratinocytes normally takes 3-4 week to grow a thin sheet of skin. Cultured keratinocytes would overcome this problem and raise the alluring prospect of banks of frozen skin readily available for transplantation. A consideration of how alloantigens are recognized in the skin suggests that cultured keratinocytes could survive without rejection.[24]

In the skin, the Langerhans cell (LC) is the dendritic cell and the immunogenicity of skin allograft correlate directly with the density of the LCs[25], which they contain functionally. However, LCs are complex because the efficiency with which they process and present antigen and activate T cell depends upon their location and differentiation.[25,26] LCs in the epidermis encounter antigen, which is than processed and displayed in association with major histocompatibility complex(MHC) class 1 or 2.[27] LCs then migrate to lymph node and mature to a cocktail of cytokine signals. In the lymph node, the MHC antigen complex displayed at the LC plasma membrane is presented to any T cell reactive to the original antigen. In skin allograft responses, the evidence is that host T cells are activated by donor LCs which migrates from the graft to the draining lymph node. Thus if the afferent lymphatics are severed, graft rejection is either abrogated (Barker and Billingham[28], 1968) or delayed (Tilney and Gowans[29], 1970).

Langerhans cells are nonadherent *in vitro* and are eliminated from primary keratinocyte culture within 7 days as a consequence of medium changing (Morhenn et al[30], 1982; Hammond et al[31], 1987). Cultured keratinocyte do not constitutively express MHC class 2 glycoproteins or the co-stimulatory molecules CD80 CD86 (Nickoloff and Turka[32], 1994) and fail to stimulate autologous lymphocyte in mixed lymphocyte reactions.

There is very little clinical or histological evidence for acute rejection of cultured keratinocyte allografts on ulcers or wounds.[33-35] Aubock et al[36] (1988) used cultured allograft to partially cover tangentially excised third degree burns and split thickness donor site. These grafts were rejected after 14 days, about 4-5 days longer than that reported for split thickness allograft. Histologically, rejection was accompanied by a mononuclear cell infiltration and vacuolation and necrosis of grafted epithelium.[24,27,35,37,38]

THE PRESENT AND FUTURE OF SKIN ALLOGRAFT

There is now a good understanding of the multiple mechanisms of allograft rejection. The hope of inducing graft acceptance persists and seems more likely in the future. Unlike transplantation of other organs, skin is readily available in the patient himself/herself and has lowest morbidity in the live donor.[9,14,33,34] For some other organs, only dead can donate.

The use of chronic immunosuppression, with associated toxicity, for prolonged survival of skin allografts is not justified. Instead some form of site specific immunosuppression is being investigated, where the immunosuppressant is delivered to the site of allograft application without any systemic administration.

Skin transplant, where the outcome is complete regeneration of the morphologically normal organ, remains both an experimental and a clinical challenge. Our understanding of the role of the skin in the immune responses and the modulation of these responses has advanced significantly.

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Chapter

9

Microbiology in Burns

Deepthi Nair

INTRODUCTION

Burns wound injuries continue to be a prevalent and devastating form of trauma. These patients require immediate and effective care in order to minimize morbidity and mortality. The survival rates for burn patients have improved substantially in the past few decades due to advances in modern medical care. The improved outcome has been attributed to advances in fluid therapy, nutrition, pulmonary care, burn wound care and infection control practices. As a result, burn-related deaths have been halved within the past 40 years.[1-3] In patients with severe burns with more than 40% of the total body surface area (TBSA) involvement, 75% of all deaths continue to be due to sepsis from burn wound infection and other infectious complications which are potentiated immensely by inhalation injury.[4-6]

Burn wound infections have been enigmatic in terms of diagnosis. Multiple factors contribute towards this fact, which include recognition of colonization versus true infection, numerous/multiple pathogens/colonizers, confounding symptoms and signs, poor sampling methods and difficult microbiological techniques.[7] Several workers have published innumerable papers on evaluating the most accurate and reproducible methods for diagnosing wound infections. Chronic burn wounds contain a large number of bacteria inherent within them. The numbers of bacteria which constitute infection in an ordinary acute surgical wound (i.e., > or = 10^5 microorganisms/gm of tissue) are not valid in burn wounds. Direct correlation with quantitative count is now seen not to be a fool proof method. Deep fluid culture correlated with histopathology examination and clinical signs and symptoms, in immunocompromised patients like burns has been found to be more useful than quantitative counts alone. Despite levels of microorganisms as great as 10^8/gm of burned tissue, these burn wounds go on to heal adequately.[8-10] Burn wounds often contain three or more microorganisms, both Gram-positive and -negative bacteria, as well as aerobic and anaerobic species. They can often contain fungal flora especially in long standing wounds where multiple broad spectrum antibiotics have been given. This cohabitation of flora with the resultant competition for nutrients and space is thought to decrease their virulence and ability to harm tissues.[8,11] Due to differences in the way acute and chronic wounds respond to diverse numbers of microorganisms, emphasis is currently being placed on clinical signs and symptoms as compared to actual numbers of microorganisms in a wound.

Microorganisms invade the burn eschar over time leading to burn wound infection and progressing to burn wound sepsis. The use of systemic antibiotics, availability of effective topical treatments and early burn wound excision with closure have all decreased the incidence of burn wound sepsis. Along with the classical signs of infection (erythema, edema, heat, purulent exudates and pain) there are a few more criteria which have been included, viz., serous exudates with concurrent inflammation, delayed healing, discoloration of granulation tissue, friable granulation tissue, pocketing at the base of the wound, foul odor and wound breakdown. As the patient is being monitored the symptom of increasing pain and dehiscence of wound is a good predictor of infection.[11,12] There are several sequels of burn wound infections which include cellulitis, septicemia, gas gangrene, graft failure, delayed healing, scarring,

IMMUNOLOGY OF SKIN

The thickness of the epidermis and dermis vary with the site of the body, sex, age of patient, etc. Infants, young children, and elderly adults have a thinner dermal layer of their skin, resulting in deeper burn injury. The dermis is capable of producing new epithelial cells to replace those lost from the epidermis by the shafts of appendages which are lined with epithelial cells. Nerve endings occur throughout both skin layers and the connective tissue of the dermis also provides a firm structural base for the skin to renovate.[3] Immunologically, thermal injuries cause an immunocompromised state. This was evident as homografts are seen to survive well in these patients.[14, 15] Host defenses can be divided into innate and adaptive immune responses. The innate immune response is the first line of defense acting promptly after the breach of skin by the burn wound.[16] When the wound covers large regions or critical organ systems or is deep, the systemic component of immunity also is evoked. The response results in a varied spectrum of presentation, with serious complications like systemic inflammatory response syndrome (SIRS), severe sepsis and multiple-organ dysfunction syndrome (MODS) which result in increased morbidity and mortality.[17]

The body tries to maintain homeostasis by a process of contraction, retraction, and coagulation of blood vessels immediately after a burn. Three zones have been described within the wound:

i. Zone of coagulation, which comprises the dead tissues that form the burn eschar that is located at the center of the wound
ii. Zone of stasis, which comprises tissues adjacent to the area of burn necrosis that is still viable but at risk for ongoing ischemic damage due to decreased perfusion, and
iii. Zone of hyperemia, which comprises normal skin with minimal cellular injury that has predominant vasodilatation and increased blood flow as a response to injury.

The burn wound surface (in deep partial-thickness and in full-thickness burns) is a protein-rich environment consisting of avascular necrotic tissue (eschar) that provides a favorable niche for microbial colonization and proliferation. The lack of vascularity leads to an impaired migration of [illegible] released by eschar tissue further [illegible] responses. Although burn wound surfaces are sterile immediately following thermal injury, these wounds eventually become colonized with microorganisms.[18-22]

MICROBIAL ETIOLOGY

Microorganisms colonizing the burn wound originate from the patient's endogenous skin, gastrointestinal and upper respiratory tracts. The normal microflora of the gut, oral cavity and vagina are both diverse and abundant. Microorganisms may also be transferred to a patient's skin surface via contact with contaminated external environmental surfaces, water, fomites, air and the soiled hands of health care workers.[23] It is the slow healing wounds which have continued exposure of devitalized tissue which allow for colonization.

Immediately following injury, Gram-positive bacteria from the patient's endogenous skin flora or the external environment are the initial culprits.[22] Endogenous Gram-negative bacteria from the patient's gastrointestinal flora also rapidly colonize the burn wound surface in the first few days after injury.[24] Wounds with a sufficiently hypoxic environment are susceptible to colonization by a wide variety of endogenous anaerobic bacteria. In most published studies, the cultures for anaerobic bacteria are minimal or omitted. However, when wounds are investigated by appropriate microbiological techniques, anaerobes are found in large numbers. Anaerobes have been termed "the secret pathogens" and "invisible villains". Wound colonization by yeasts and fungi usually occurs later due to the use of broad-spectrum antibiotic therapy.[25] Microorganisms transmitted from the hospital environment tend to be more resistant to antimicrobial agents than those originating from the patient's normal flora.[26] Tables 9.1 and 9.2 list the most common microorganisms colonizing and infecting burn wounds. Prior to the antibiotic era, *Streptococcus pyogenes* (Group A beta-hemolytic streptococci) was the predominant pathogen implicated in burn wound infections and a major cause of death. In 1950's, with the introduction of Penicillin G, Streptococci were 'wiped off' and replaced by *Staphylococcus aureus*. Even today, *Staphylococcus aureus* remains a common cause of early burn wound infection. The role of *Pseudomonas aeruginosa* from the patient's endogenous gastrointestinal flora and/or an environmental source is one of the foremost causes of burn wound

Table 9.1: Bacteria and fungi that constituted burn wound infection: National Nosocomial Infections Study of the Center for Disease Control, USA

Species	*Percentage of isolates*	
	(1970-74)	*(1980-98)*
Staphylococcus aureus	22.9	23
Pseudomonas aeruginosa	20.9	19.3
Pseudomonas species	7.2	
Escherichia coli	6.7	7.2
Group D streptococci	5.0	11
Streptococcus faecalis	4.2	
Klebsiella pneumoniae	3.7	2.6
Serratia marcescens	3.1	3.5
Enterobacter cloacae	3.0	9.6
Proteus mirabilis	2.8	
Enterobacter species	2.5	
Klebsiella species	2.2	
Staphylococcus epidermidis	1.4	4.3
Group A streptococci	1.1	
Enterobacter aerogenes	1.0	
Candida albicans	1.3	3.5
Others		16

infections in many centers.[27] The incidence of rare infective pathogens has also increased steadily[28] (Table 9.2). While less common, infections due to anaerobic bacteria typically occur secondary to electrical burns or when open wound dressings are used in place of occlusive dressings.[29]

The worldwide emergence of antimicrobial resistance among a wide variety of bacterial and fungal pathogens, particularly nosocomial isolates, limits the available therapeutic options for effective treatment.[27] MRSA, methicillin-resistant *Staphylococcus aureus*, Vancomycin-resistant enterococci and multiple resistant gram-negative bacteria that possess several types of beta-lactamases, including extended spectrum beta-lactamases, AmpC beta-lactamases and metallobeta-lactamases have been emerging as serious pathogens in hospitalized patients.[26] Fungal pathogens, particularly *Candida* spp., have increasingly become important opportunistic pathogens due to the use of broad-spectrum topical and systemic agents when infection occurs in the burned patient and have demonstrated increasing degrees of antifungal drug resistance. [30]

Table 9.2: Microflora of wounds

Aerobic and facultative microorganisms found and the Type of wound (A-Acute, C- Chronic)

Gram-positive:

- Coagulase-negative staphylococci A, C
- *Staphylococcus aureus* A, C
- Beta-hemolytic *Streptococcus* (group C) A, Beta-hemolytic *Streptococcus* (group G) C
- *Streptococcus* spp. (fecal) A,
- *Streptococcus* spp. (viridans) A, C
- *Streptococcus intermedius* C
- *Corynebacterium xerosis* C, *Corynebacterium* sp. A, C
- *Micrococcus sp.* C
- *Bacillus sp.* A

Gram-Negative:

- *Escherichia coli* A, C *Escherichia hermanii* A
- Serratia liquefaciens C
- *Klebsiella pneumoniae* A, C, *Klebsiella oxytoca* A, C
- *Enterobacter cloacae* A, C, *Enterobacter aerogenes* C
- *Citrobacter freundii* C
- *Proteus mirabilis* A, C,*Proteus vulgaris* C
- *Providencia stuartii* A
- *Morganella morganii* C
- *Acinetobacter calcoaceticus* A, C
- *Pseudomonas aeruginosa* A, C
- *Stenotrophomonas maltophilia* A

Anaerobic and facultative anaerobic microorganisms isolated from acute and chronic wounds of varied etiology

- *Clostridium limosum* A *Clostridium bifermentans* A *Clostridium difficile* C *Clostridium sporogenes* A, C Clostridium ramosum C Clostridium tertium A *Clostridium histolyticum* A, C *Clostridium septicum* A *Clostridium baratii* C *Clostridium clostridioforme* A, C *Clostridium perfringens* A, C *Clostridium cadaveris* A, C
- *Peptostreptococcus asaccharolyticus* A, C *Peptostreptococcus anaerobius* A, C *Peptostreptococcus* sp. A, C *Peptostreptococcus micros* A, C *Peptostreptococcus prevotii* A, C *Peptostreptococcus magnus* A, C C *Peptostreptococcus indolicus* C
- *Bacteroides thetaiotaomicron* C *Bacteroides caccae* C Bacteroides capillosus C Bacteroides stercoris C *Bacteroides fragilis* A, C *Bacteroides uniformis* A, C *Bacteroides ovatus* A *Bacteroides ureolyticus* A, C
- *Prevotella oralis* A, C *Prevotella oris* A, C*Prevotella* disiens A*Prevotella bivia* C *Prevotella buccae* C*Prevotella* sp. A *Prevotella corporis* A, C*Prevotella intermedia* A *Prevotella melaninogenica* C
- *Porphyromonas asaccharolytica* A, C
- *Propionibacterium acnes* A, C
- *Sphingobacterium multivorum* C
- *Eubacterium limosum* C
- *Fusobacterium necrophorum* C
- *Veillonella spp.* A

Fungi

- *Candida parapsilosis* A,
- *Candida krusei* A

QUANTITATIVE MICROBIOLOGY: SIGNIFICANCE OF MICROBIAL NUMBERS

The clinical significance of the microbial load in delaying wound healing was described in 1964 by Bendy et al[31] who swabbed decubitus ulcers and discovered that bacterial load of 10^6 CFU/ml of wound fluid delayed healing. Later tissue biopsy specimens were demonstrated to be superior samples.[32] Quantitative culture of tissue biopsy specimens is demanding on the microbiology laboratory and of minimal value in facilitating prompt burn wound management. It has been observed that a rapid Gram stain can reliably predict a microbial load of 10^5 CFU/g of tissue if a single microorganism was seen on the slide preparation.[33,34] More recently it has been[35] demonstrated that a critical level of bacteria of $>10^4$ CFU/g of tissue must be reached to cause infection in wounds and that quantitative tissue cultures can predict the likelihood of wound infection more effectively than swab cultures. Some authors prioritize histopathology over all other methods as it demonstrates true invasion. [36] Another technique which has been utilized is the velvet pad surface imprint technique. Dermabrasion as a sampling method has also been used.

The two most popular methods have been quantitative studies on the basis of surface sampling techniques and deep-tissue biopsy techniques. The value of superficial cultures in wound assessment has been questioned and it is stated that purulent wound fluid may fail to yield growth whereas biopsied tissue may yield significant numbers of bacteria.[37] It has also been stated that superficial tissue is likely to harbor a diversity of aerobic and anaerobic microorganisms, one or more of which may invade deeper tissue. A situation where the superficial tissue is "sterile" while deeper tissue is "infected" is very improbable. Most wounds are colonized with microorganisms and a failure to isolate them is more likely to be a consequence of poor microbiological technique. Some workers have stated that[38] quantitative bacteriology for burn wounds provides little information beyond that obtained from a surface swab and swabs that yield more than 30 CFU reliably indicate a tissue count of 10^5 CFU/g. The need to quantify bacteria by performing tissue biopsy, which is invasive, potentially traumatic to the patient and non-routine, requiring expert manipulation and creates an increased workload for the microbiology laboratory, is debatable. The role of tissue biopsies has retained significance in detection of fungi, anaerobes and other relatively rare pathogens.

SIGNIFICANCE OF SPECIFIC MICROORGANISMS

The role of specific types of microorganisms on progression and healing of burn wounds has been studied extensively. Presence of microorganisms and their identity as the etiological agent in infection or delayed healing can be confirmed only if they are present as a pure mono-microbial flora. Burn wounds tend to be of polymicrobial etiology involving both aerobes and anaerobes. It is not possible to differentiate between pathogenic and nonpathogenic species in polymicrobially infected wounds and the presence of a pathogen in a mixed-culture infection does not necessarily establish that particular microorganism as the etiologic agen The diagnosis of infection in polymicrobially infected woun should be based primarily on clinical signs, such as hea pain, erythema, edema, suppuration and fever; microbi logical results may be helpful but can often be misleadin However, when clinical signs of infection are less eviden greater emphasis may have to be placed on microbiologic results. Aerobic pathogens such as *S. aureus*, *P. aeruginos* and Gram-negative bacteria etc. have been frequentl implicated as the cause of delayed wound healing an infection. *S. aureus* is considered to be the most problemati bacterium in burn wound infections, primarily based on th knowledge that its incidence is high and complications ten to be higher.[39, 40] A consensus was reached in a meeting the European Tissue Repair Society and the European Woun Management Association that the presence of beta-hemolyti (group A) streptococci or *P. aeruginosa* in a chronic woun was an urgent indicator for initiating antimicrobi therapy.[41,42]

A symbiotic environment develops in long standing bur wounds as observed in the association of aerobic an anaerobic microorganisms. It has been observed that (oxygen consumption by aerobic bacteria induces tissu hypoxia and a lowering of the redox potential, which favo the growth of anaerobic bacteria, (ii) specific nutrien produced by one bacterium may encourage the growth fastidious and potentially pathogenic cohabitin microorganisms. Thus, *S. aureus* promotes the growth of vitamin K-dependent strain of *Prevotella melaninogenic* *in vitro*[43] and the pathogenicity of *P. melaninogenica* h been related to the presence of vitamin K. *K. pneumonia* was reported to be instrumental in enhancing virulence i *P. melaninogenica* by providing succinate as an essenti growth factor, and (iii) some anaerobes are able to impa host immune cell function and thus provide a competiti advantage to themselves and other cohabitin microorganisms. Antiphagocytic activity in *melaninogenica* was expressed only in the presence of hemi and vitamin K provided by microbes like *S.aureus*. Succina also inhibits the chemotaxis and phagocytosis of *E. co* and *S.aureus*.

In theory, the presence of *S. aureus* and a pigmenti gram-negative anaerobe in a wound is likely to be mo detrimental than the presence of a similar quantity of *aureus* alone. Therefore, the composition of t polymicrobial wound flora is likely to be more importa than the presence of specific pathogens, since this w determine whether pathogenic interactions are likely to occur. Another critical factor in wound healing which cannot be underestimated is immune response in dealing with wound micro flora. Factors such as tissue necrosis, hypoxia and ischemia impair immune cell activity in a wound, as do debilitating conditions like diabetes mellitus, chronic granulomatous disease, other immune deficiencies and microorganisms can also participate in compromising the immune response. Only by assessing the host and microbial factors collectively, along with symptomatology and clinical signs of the patient can the problem of burn wound infection be addressed.

BIOLOGICAL ANALYSIS OF WOUNDS

There is little consensus whether sampling is relevant, when and how a wound should be sampled, how a specimen should be transported to the laboratory and what analyses should be requested. Confusion also exists in view of the fact that health care practitioners often consider a microbiological report to provide definitive information on whether a wound is infected [44] and the provision of an antibiogram for a particular pathogen can often be misleading and prompt unnecessary treatment. However, having stated this it still remains critical that the burn wounds be assessed clinically, microbiologically and histopathologically as stated earlier in this chapter. It can predict worsening infection, internal spread and the data generated is extremely vital in terms of hospital infection control.

Wound-sampling Methods

In generalized terms, the quantitative and qualitative microbiology of wounds can be investigated by sampling either wound tissue or wound fluid. The fluid is usually swabbed and the tissue biopsies are performed.

Wound Tissue Sampling

The acquisition of deep tissue biopsy following initial debridement and cleansing of superficial debris is the most useful method for determining the microbial load and the presence of invasive pathogens. Tissue is obtained aseptically and processed by weighing, homogenizing, serially diluting and plating onto selective and nonselective media. These are then incubated under aerobic and anaerobic conditions to provide quantitative and qualitative information. Superficial, devitalized tissue removed by curettage, may also be investigated for microbial content. Another technique involving dermabrasion is less invasive as compared to the biopsy.[45] Multiple samples from several areas of the burn wound should be obtained and sent for workup. After cleaning the wound with isopropyl alcohol, 2 parallel incisions 1-2 cm in length and 1.5 cm apart with a depth to obtain a portion of the underlying fat are made in the skin. These are similar to split skin smears. Biopsies 0.02-0.5 g may be obtained with a 3 mm punch-biopsy technique. This is a technically demanding technique.

Histopathological evaluation can detect bacteria, fungus (periodic acid-Schiff [PAS] and Gomori methenamine silver [GMS]), although cultures must be obtained to definitively identify and give sensitivity of the pathogen. Viruses like the Herpes simplex virus (Giemsa) can be associated with wounds and are isolated via identification of inclusions on light or electron microscopy. Histology also guides in the staging of the burn wounds.[46,47] The various stages used to diagnose burn wound infections are as follows:

- Stage I—Colonization
    a. Superficial—Microorganisms present only on burn wound surface
    b. Penetrating—Variable depth of microbial penetration of eschar
    c. Proliferating—Variable level of microbial proliferation at nonviable–viable tissue interface (subeschar space)
- Stage II—Invasion
    a. Micro-invasion—Microorganisms present in viable tissue immediately subjacent to subeschar space
- Stage III—Deep invasion
    a. Penetration of microorganisms to variable depth and expanse within viable subcutaneous tissues.
    b. Micro-vascular involvement: Microorganisms within small blood vessels and lymphatics (thrombosis of vessels is common).

Wound Fluid Sampling

Wound swabs are taken using cotton-tipped swabs to sample superficial wound fluid and tissue-debris. When a copious volume of burn wound fluid exists, sampling by needle aspiration can be employed. This is superior to swabbing. The syringe technique may be used to sample deeper pockets of fluid. If the technique is performed with strict asepsis samples can be retrieved without any significant exogenous contamination. In pus containing cavity, irrigation with sterile saline and gentle massaging may be performed to provide fluid for aspiration.

An alginate-tipped swab can also be used to perform a fully quantitative analysis, since the swab will dissolve and release all associated microorganisms when transferred to an appropriate diluent. Despite its widespread use, there is debate over the value of the swab sampling technique and the value of cleansing a wound before swabbing is performed. A variety of other techniques, including the dry and presoaked velvet pad, filter paper disks and cylinder scrubbing, have also been used to sample superficial wound fluid for microbiological analysis. The immense number of sampling methods available creates a problem since all are reputed to have benefits and there is no single, universally accepted method. Thus, the debate and controversy continues regarding the type of sample required to provide the most meaningful data.[41,45,48]

The results generated by the microbiology laboratory may be misinterpreted and encourage inappropriate treatment. Without question, tissue biopsy or excision is the gold standard for microbial conclusive evidence. This method is most beneficial in determining the optimal time for skin grafting and surgical wound closure.[49] However, the value of a single biopsy specimen, particularly in chronic wounds, is debatable. Particularly in slow-healing chronic wounds that require long-term care, tissue biopsy is of minimal value since it requires expert surgical technique, is non-routine and is potentially traumatic to the patient. Aspirates of purulent fluid are excellent representatives of the wound infection. The role of superficial swab samples has been intensely debated but the procedure is simple, inexpensive, noninvasive and convenient for the majority of wounds. Swab sampling has been challenged on the basis that the superficial colonizers do not reflect the organisms of deeper tissue and that subsequent cultures do not correlate with the presence of pathogenic bacteria.[48] The collection of the swab should be meticulous, if taken prior to wound cleansing or the removal of devitalized superficial debris, the resulting culture will only have superficial colonizers and provide misleading or useless information. Vital tissue must be sampled or a specimen should not be collected at all and the patient should be treated empirically.[50]

Another theory has it that since the majority of wounds are contaminated with endogenous microorganisms, any flora present in deeper tissues are also likely to be present in the superficial debris. Thus, there are arguments to support the use of the swab sample as a useful method. For routine management, perhaps it can be suggested that only wounds that are clinically infected or those that have no clinical signs of infection but are deteriorating or have a long his of failure to heal should be sampled for microbiolog analysis. However, indiscriminate and inappropr swabbing of wounds causes an unnecessary drain on la and financial resources. An important factor to consi when sampling a wound for microorganisms is administration and route of antimicrobial therapy. If a pat is receiving treatment, microbial isolation from swab samp is likely to be significantly influenced by topical antimicrob agents whereas the microflora of deep tissues is more lik to be influenced by systemic antibiotic therapy.

In summary, superficial swabbing can be justified a simple procedure for assessing the microflora of wou that are clinically infected or failing to heal. Semi-quantitat analysis will provide a good indication of the microbial lo and the qualitative analysis, although complex will prov an indication of the diversity of microorganisms and poten for microbial synergy. A request for both aerobic a anaerobic microbiological investigation is an import aspect of obtaining the information required to gui appropriate antibiotic therapy.

Sample Transport

The prompt delivery of the specimen to the laboratory considered to be of utmost importance. This is particular true for swabs which are susceptible to drying up. Aspirat of purulent fluid and tissue samples are preferable to swa in these circumstances because they maintain the conditio required to sustain microbial viability, i.e. a moist and reduc environment. There are prereduced commercially availab transport media which can be used if culture is delay beyond 1 to 2 hour after sampling. This offers a cheap an effective method to enable the culture of both aerobic an anaerobic microorganisms. Since swab samples a susceptible to desiccation and oxygen exposure, prereduced, nonnutritive transport medium is essential t maintain the viability of microorganisms on cotton swab Storage at room temperature, 25°C, is considered to b appropriate for the maintenance of most microorganism temperatures above or below this may cause inhibition o death of some flora.

Processing of Wound Specimens

On arrival at the laboratory, a specimen must be accompanie by a clinical description of the burn wound. Informatio regarding the type/degree of wound, the position of th wound, clinical signs of infection, presence of necrosis, associated malodor, and antimicrobial therapy. All these greatly assist the microbiologist in appropriate selection of tests and accurate interpretation of the results of the laboratory data. Also, the provision of information regarding current antibiotic treatment may aid microbiologist in determining which antimicrobials have become less useful for the microorganisms. Microbial culture and antibiotic sensitivity results are usually generated in about 48-72 hours (and may, on occasion, take considerably longer) when using manual methods.

A number of rapid investigations must be considered. Direct Gram stain of the swab/pus/biopsy is one of the most reasonable and effective rapid tests in the microbiology department which has stood the test of time, for over a century, Gram's stain is still the most important stain in microbiology.[52] It is widely used as a rapid technique for guiding antibiotic therapy in life-threatening infections (Figs 9.1 and 9.2). Also, the presence of microorganisms in a Gram stained smear prepared from a wound swab has been shown to consistently reflect a microbial load of $>10^6$ organisms isolated by a quantitative swab technique from open burn wounds. Speciation on the basis of Gram's stain is possible only for gram-positive spore-forming anaerobes such as *Clostridium perfringens*. However, the value of the Gram stain as a final diagnostic tool is debatable as contaminant, colonizer and true pathogen cannot be differentiated. Its use in the evaluation of samples with risk of contamination with skin/mucosal micro flora has been reported as insufficiently reliable to guide antibiotic selection. Gram staining of tissue biopsy specimen homogenate has often been used to rapidly estimate the microbial load.[53] The combined presence of leukocytes and bacteria is likely to be a good indicator of infection.[55] Having said that, Gram stain remains a reliable, cheap, easily available and standardized rapid method to diagnose wound infections, particularly with single pathogen.

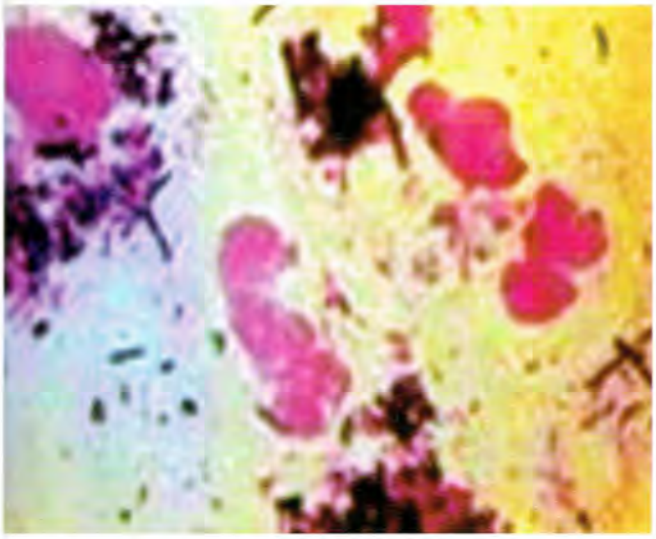

Fig. 9.1: Gram stain of pus from wound showing polymorphs and gram-positive and negative bacteria

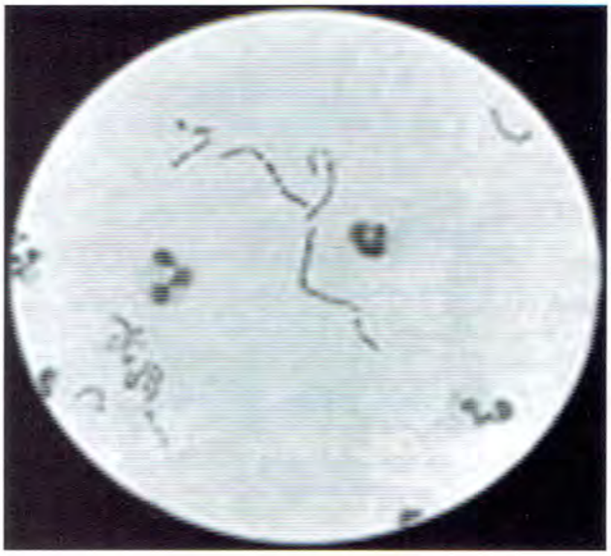

Fig. 9.2: Pus cells with streptococci in chains

Other stains which have potential in assessing burn wounds are the fungal stains. Potassium Hydroxide (KOH) is used to dissolve keratin and highlight fungal elements in wet smears. It has a great role to play in nail, hair and skin samples, although the role in burn wounds is limited. Lactophenol Cotton blue preparation is an excellent and cheap stain used for staining and fixing fungal elements, which are very clearly delineated using this method. Histopathological tissue can be fixed and stained with Periodic acid Schiff's base (PAS) which specifically stains and demarcates the only fungal tissue. More expensive stains as the fluorescent Calcuofluor white stain, immunoperoxidase and other immunohistochemical stains are used in specialized set-up. Giemsa has been used successfully to demonstrate inclusion bodies of viruses like herpes and also in mycology.

Culture and Sensitivity of Wound Specimens

Routine isolation analysis of wound specimens normally involves the use of selective and nonselective media to culture aerobic, anaerobic bacteria and yeasts (Fig. 9.3). If a

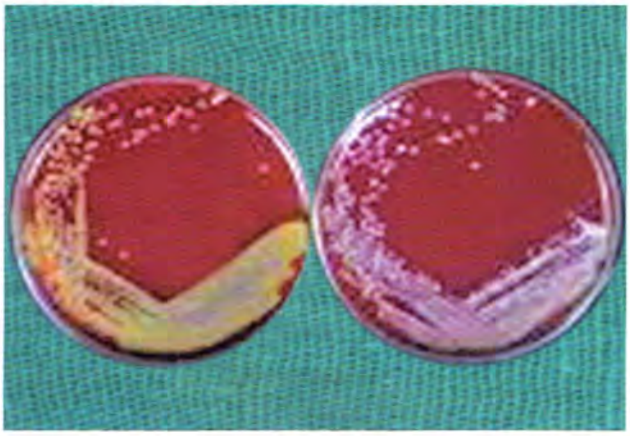

Fig. 9.3: Blood agar plates showing growth of *Staphylococcus aureus*

specimen is purulent and/or malodorous, anaerobic bacteria also must be suspected, anaerobic microbiology is often excluded from a routine analysis. Following incubation under aerobic or anaerobic conditions for 24 to 48 hours, qualitative and semi-quantitative assessments of the cultures are normally made. *S. aureus*, *P. aeruginosa*, and beta-hemolytic streptococci are generally singled out as potential aerobic pathogens and their growth is often reported as being light to profuse.[56] With the exception of *Clostridium* spp., anaerobes (if investigated) are likely to be reported as being "mixed" with aerobic micro flora. Antibiograms are most frequently performed for the aerobic pathogens, particularly if they are cultured in abundance and with minimal cohabiting micro flora. If aerobes are absent but the wound is reported as being clinically infected, anaerobes should be suspected and investigated. Many of the endogenous anaerobic bacteria require between 2 and 5 days to grow on selective culture media However, in mixed aerobic-anaerobic cultures (Fig. 9.4) that reflect polymicrobial wounds, the fastidious anaerobes often grow more readily due to the availability of essential growth factors provided by cohabiting facultative bacteria. The following criteria give rough guidelines to assess microscopy and culture of burn wounds—

i. A Gram stain should always be performed. If numerous leukocytes are observed, together with a single bacterial morphology an early indication of the probable pathogen can be provided.
ii. Wound specimens should be cultured for aerobic, anaerobic and fungal microorganisms.
iii. At least one selective and one non-selective culture medium should be used for the isolation.
iv. If one microorganism is clearly prevalent (i.e., in p culture or in abundance with minimal involvemen other microorganisms), an antibiogram should performed.
v. Antibiograms should also be performed on preval microorganisms in mixed cultures, particularly if la numbers of leukocytes are observed in the origi Gram stain of the specimen.

Burn centers should routinely determine and track specific pattern of burn wound microbial colonization, ti related changes in the predominant microbial flora of burn wound in individual patients, the antimicrob susceptibility profiles of microorganisms implicated in b wound infections in a given time period and trends in nosocomial spread of these pathogens. Some burn cent rely on a laboratory microbiologic surveillance syst involving periodic sampling of burn wounds and ot anatomic sites to provide this information.[7]

Microorganisms within a complex burn wound biofi are in a physiologically and metabolically different state fr their *in vitro* planktonic or free living forms. There currently no published standards for the routine antibi susceptibility testing of bacteria that form part of a biofil Modified broth micro-dilution plate method may provide

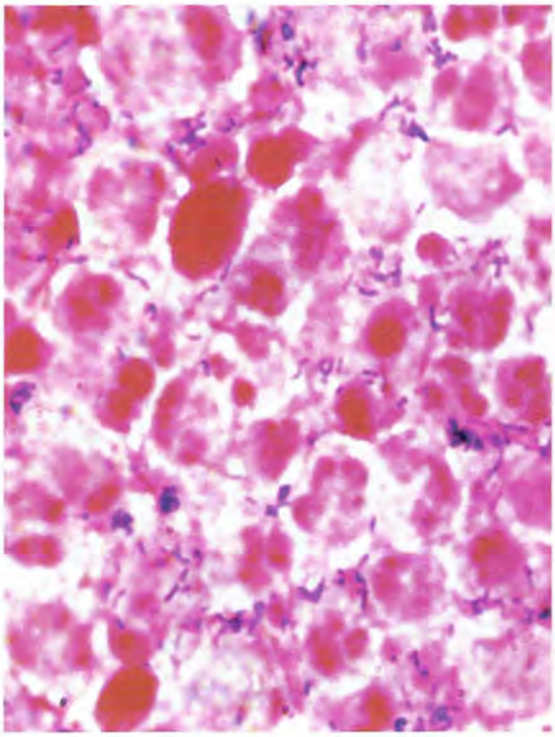

Fig. 9.4: Mixed aerobic and anaerobic flora in burn

accurate result. Infection control practices should be at their apex in burn units. The following of strict infection control practices of physical isolation in separate room, use of gowns and gloves during patient contact, hand washing before and after each patient visit and appropriate empirical antimicrobial therapy are the critical needs. These greatly aid in reducing the incidence of infections due to antibiotic resistant organisms. An institutional policy guiding appropriate selection and use of antimicrobials for the treatment of infections in burn patients further support efforts to reduce the burden of illness due to antibiotic resistant organisms while potentially reducing hospital costs, length of hospital stay, and adverse effects due to these agents.

Reporting of Microbiological Results

The laboratory has a key role to play in providing information about wounds that are deteriorating or failing to heal. From a microbiological perspective, the main pathogens or groups of microorganisms that the microbiology laboratory should routinely detect and report (with antibiograms being provided when appropriate) are as follows: *S. aureus*, *P. aeruginosa*, beta-hemolytic streptococci, coliform bacteria, pigmented gram-negative anaerobes (*Prevotella* and*Porphyromonas* spp.), non-pigmented gram-negative anaerobes(primarily *Bacteroides*, *Prevotella*, and *Fusobacterium* spp.), *Peptostreptococcus* spp., and *Clostridium* spp. Fungal species like *Candida* spp, *Aspergillus*, Mucor, *Penicillium* etc. must be sought in patients with long standing burns having broad spectrum antimicrobial therapy. In order that the microbiology laboratory can provide the wound care practitioner with clinically relevant information, it is essential that the microbiology results (e.g., Gram stain, culture, and antibiograms) be interpreted in association with clinical information provided by the practitioner (e.g., wound type, location, condition, signs of infection, and sampling method). In this respect, the medical microbiologist may need to interpret the significance of a particular wound pathogen in the light of the clinical presentation.

In summary, a continuous dialogue between the microbiology department and the clinician is essential to ensure that:

i. Only those burn wounds that are likely to benefit from a microbiological investigation are sampled (i.e., those with clinical signs of infection or those that are failing to heal);
ii. The microbiologist has a thorough understanding of the clinical presentation of the wound which is possible when the laboratory forms are filled with adequate clinical information;
iii. The microbiologist must know the method used for wound sampling;
iv. The microbiologist is aware of the requirements of the practitioner and the urgency of the results and
v. The practitioner understands the rationale for advice given by the microbiologist, e.g. the reporting of a mixed culture and an antibiogram for a bacterium isolated from a mixed culture may not be provided if clinical signs of infection are not evident and if no inflammatory cells are seen in the Gram stain.

This interaction between clinician and microbiologist in the management of burn wound complications definitely results in significant savings in cost and time (medical, nursing and microbiological) along with an appropriate treatment for the patient.

OTHER MICROBIOLOGICAL METHODS

Blood Cultures—Blood cultures are essential in determining septic episodes. The best time to collect the specimen is before the temperature spikes. A temperature above 39.5°C is the body's method for cleansing the blood stream of bacteria. Most bacteria do not survive at prolonged temperature above 39.5°C. Care must be taken not to contaminate the blood culture bottle. Site selection for collecting the specimen must be meticulously and aseptically cleansed prior to specimen collection. If the site is through a contaminated area, appropriate comments should be made on the request slip. These considerations are somewhat aberrant in the burn patients as the body temperature may not represent sepsis and even acquiring samples for blood culture is difficult in extensively burned patients.

Diagnosis of sepsis in burn patients can be difficult to distinguish from the usual hyperdynamic, hyperthermic, hypermetabolic post-burn state. Blood cultures are commonly negative. Fever spikes are not proportional to degree of infection.

Clinical diagnosis of sepsis is made by meeting of at least 3 of the following criteria:

1. Burn wound infection ($>10^5$ organisms/gm tissue) with histologic or clinical evidence of invasion
2. Thrombocytopenia (<50,000) or falling rapidly
3. Leukocytosis or -penia (>20,000 or <3,000)
4. Unexplained hypoxia, acidosis or hyper/hypoglycemia
5. Prolonged paralytic ileus
6. Hyper/hypothermia (>39°C or <36.5°C),

7. Positive blood cultures
8. Documented catheter or pulmonary infection
9. Altered mental status
10. Progressive renal failure or pulmonary dysfunction

Urinary Tract Infection (UTI)—If a UTI is suspected as the cause of sepsis, urine must be collected aseptically. Appropriate comments should be made on the request slip (e.g. Temp. 39.8°C, clean catch, catheterized, suprapubic, etc.)

Respiratory Infection—If a respiratory infection is suspected or evident from clinical signs or an X-ray, bronchial washings are essential in order to identify the etiologic agent or infection. However, based on recent mortality studies, the organism isolated from lung abscesses are most closely correlated with organisms from the wound, rather than the organisms isolated from sputum specimens.

AUTOMATION IN CULTURE AND SENSITIVITY

Cultivation of the fluid or macerated tissue sample using rapid systems of isolation such as BacT Alert 3D, BACTEC (for pyogenic bacteria), TREK/ESP systems, etc. can greatly hasten (6-24 hours) the isolation of pathogens but have a limited role to play in mixed flora. However, quantifying the samples has not been standardized using these systems. They also are unable to process swabs, velvet pads, filter paper, etc. Fluid, pus or homogenized tissue is an appropriate sample for these systems. Their prohibitive cost precludes their use in routine and also in surveillance. These automated machines have a critical role to play in blood cultures in burn wound septicemias.[7]

Most laboratories now routinely use an automated system for identification and susceptibility testing of the bacteria. These are like the VITEK from Bio-Me´rieux, Durham, NC; Phoenix from Becton Dickinson, Franklin Lakes, NJ; or Microscan from Dade-Behring, Deerfield, IL, etc. These must be in combination with disk diffusion for screening and E-test, and agar dilution for confirmation of some of the automated system's susceptibility results. This is particularly true when unusual multiple drug resistant organisms (MDRO) are observed like Vancomycin intermediate or resistant *Staph. aureus* (VISA/VRSA), Vancomycin resistant *Enterococcus* (VRE), MDR *Acinetobacter* spp and MDR *Pseudomonas* spp.

Most antimicrobial agents have not been subjected to controlled clinical efficacy trials in the treatment of burn wound infections and there are limited data about the ability of systemically administered antibiotics to penetrate the devascularized burn eschar.[59] Hence, burn patients infect with bacterial strains that appear to be susceptible to particular antimicrobial agent *in vitro* may fail to respo clinically. These apart, standard methods have not yet be published to allow clinical microbiology laboratories routinely perform antibiotic susceptibility testing of bu wound isolates against commonly used topical antimicrobi agents.

Polymerase Chain Reaction

The role of molecular tests in rapid diagnosis of infectiv conditions has been very well elucidated for slow growin and difficult to grow pathogens. Its role in certain infectiv diseases like tuberculosis and meningitis is noncontrovertia Detection of role of viral infections of burn wounds ha been possible with this technique. Using PCR based viru detection and immunohistochemical staining, burn woun infection with HSV - I could be identified.[57]

A real-time polymerase chain reaction to detect an quantify *Pseudomonas aeruginosa* in wound biopsy sample has been tried. This method produced a linear quantitativ detection range of 7 logs, with a lower detection limit o 10^3 colony-forming units (CFU)/g of tissue or a few copie per reaction. The time from sample collection to result wa less than 1hour. This method has potential for rapi quantitative detection of pathogens in burn patients, enablin early and individualized treatment.[58] Although molecula methods allow rapid quantitation of individual organisms bacterial tissue biopsy culture is still necessary in order t identify and perform antibiotic susceptibility testing on the different types of bacteria present in a given sample. If a predominant resistotype is expected then molecular tests to detect the mutational type can greatly aid in the rapid diagnosis of resistance. Emerging antimicrobial resistance trends in burn wound bacterial pathogens represent a serious therapeutic challenge for clinicians caring for burn patients. Antibiotic-resistant organisms such as MRSA, vancomycin-resistant enterococci, and multiple-resistant gram-negative rods, including *Pseudomonas aeruginosa*, *Acinetobacter* spp. and various members of the family *Enterobacteriaceae* have been associated with infections of the burn wound and other anatomic sites in patients with major thermal injury, occasionally in the form of nosocomial outbreaks. Role of molecular biology in rapidly identifying mutations leading to resistance and epidemiological typing of isolates using Restriction Fragment Length Polymorphisms (RFLP), Pulse Field Gel Electrophoresis (PFGE), Multilocus Strain Typing

(MLST), etc. will definitely play an enormous role in the future in burn microbiology.[7]

Gas Liquid Chromatography (GLC)

This is particularly of use in the presence of anaerobic bacteria. Since these microorganisms can be characterized on the basis of the fatty acids produced as end products of metabolism, rapid determination of the presence of potentially pathogenic anaerobic bacteria can be performed by direct GLC analysis on the specimen. However, specimens that can benefit from this type of analysis are normally restricted to purulent drainage from enclosed abscesses. Although GLC is a valuable method for rapidly identifying anaerobic bacteria in clinical specimens, the costs and expertise involved with such equipment have prevented its use as a routine procedure in microbiology laboratories.

ANTIBACTERIAL THERAPY

Burn wound progression is complex and caused by additive effects of inadequate tissue perfusion, free radical damage, and systemic alterations in the cytokine milieu of burn patients, leading to protein denaturation and necrosis. The current mainstays of treatment include adequate fluid resuscitation, nutritional support and local wound care with an emphasis on topical antimicrobial agents and biosynthetic dressings.

For the Prevention of burn wound infection

- Topical antimicrobials which should be used according to the local recommendations.
- Systemic antibiotics are not routinely indicated except as prophylaxis before dressing changes and in order to prevent septicemia related to manipulation/surgical debridement/grafting, etc of the wound (choice of agents being determined by results of previous surveillance cultures).
- Nasal mupirocin in some cases, at the time of presentation has shown some evidence of benefit to prevent staphylococcal infections.

Topical Therapy

Topical therapy is often applied to prevent infection and to treat ongoing infections or used as an adjunct to surgical treatment and systemic antibiotics. Systemic antimicrobial agents should be directed at the underlying pathogen recovered from culture or determined empirically from the local burn unit's antibiogram while culture results are pending. Antifungal agents may also be used; however, pathogen identification is necessary in order to determine the ideal antifungal agent. Topical therapy is typically applied to prevent infection and treat infection when adequate surgical management is not possible. Wound care should be directed at thoroughly removing devitalized tissue. Currently, a number of topical agents are available to assist in microbial control of the burn wound, including silver sulfadiazine, mefenide acetate, 0.5% silver nitrate, bacitracin/polymyxin B, mupirocin, and Mycostatin. No single agent is totally effective and each has advantages and disadvantages. Almost all agents affect wound healing and increase metabolic rate. These various therapies differ in their ability to penetrate eschars, antimicrobial activities and adverse-event profiles. However, they may be associated with drug pressure, resulting in infections with resistant bacteria or fungus. A broad-spectrum surgical antimicrobial topical scrub should be used along with adequate analgesia and preemptive anxiolytic in order to permit adequate wound care.

Silver sulfadiazine 1% cream is the most commonly used topical antimicrobial agent in burns. Its antimicrobial properties are derived from the dual mechanisms of its silver and sulfa moieties (functional parts) and has a broad spectrum of antimicrobial coverage including gram-positive bacteria, most gram-negative bacteria, and some yeast forms. Some gram-negative organisms (e.g. some *Pseudomonas* species) do possess a plasmid mediated resistance. Unlike mafenide or silver nitrate, silver sulfadiazine does not hinder epithelialization, although it does hamper contraction of fibroblasts. Mafenide acetate 11.1% cream (e.g. Sulfamylon®) is one of the oldest effective topical antimicrobial agents. Mafenide has a broad spectrum of antimicrobial activity, including silver sulfadiazine-resistant *Pseudomonas* and enterococci, but reduced antifungal properties. Its exact mechanism of action is not clear, but thought to be related to its water-soluble sulfa-moiety. Mafenide cream is toxic to epithelial cells and fibroblasts. Unlike other topical agents, mafenide has good penetration through the eschar. For this reason mafenide is commonly used on dirty or infected burn wounds, electrical burns and on burned ears to prevent chondritis. Petroleum-based antimicrobial ointments such as bacitracin and/or polymyxin B are clear on application, painless and allow for easy wound observation. These agents are commonly used for treatment of facial burns, graft sites, healing donor sites and small partial-thickness burns. Povidone iodine ointment has a broad antimicrobial activity, including bacteria, fungi, and some viral forms. Mupirocin (Bactroban) has improved activity against gram-positive bacteria, especially methicillin resistant *Staph. aureus* (MRSA) and selected enteric bacteria. Mycostatin should not be combined with mafenide, however, because both become inactivated. In addition, Mycostatin 5-15 ml given orally 3 times daily reduces alimentary fungal overgrowth. This regimen has markedly decreased the incidence of *Candida* septicemia in our patients. In severely burned patients (>40% BSA), the combination of Mycostatin ointment or powder with other topical agents reduces the incidence of fungal infections.[61] Topical agents have been discussed at length in another chapter of this textbook.

Systemic Antibiotic use and its Guidelines

Antibiotics are indicated when there is evidence of pneumonia (common in inhalational burn injuries) and when there is evidence of sepsis. Large burn wounds also often warrant the need for antibiotic use systemically. Specific choice of antibiotics depends on the local surveillance information and knowledge of antibiotic resistance pattern. Antibiotic prophylaxis has also been studied in burn surgery at the time of wound manipulation, but few studies have supported the use of systemic antibiotics during acute burn surgery. Antibiotics appear to be of no value in the prophylaxis of wound infections accompanying surgery for small to moderate burns. However, a few studies have recommended surgical prophylaxis in patients with burns over more than 40% TBSA. In these circumstances, if an infection is identified, antimicrobial therapy should be directed at the pathogen recovered on culture. In the setting of invasive infection or evidence of sepsis, empiric therapy is urgently initiated. A local burn facility's antibiogram must be established to help direct empiric therapy.

There is currently no consensus on optimal empiric treatment (Table 9.3) and recommendations for empiric therapy are based on the following criteria:

- Extent and depth of the burn wound
- Age, state of hydration, creatinine clearance, seru protein levels of patient
- Presence of sepsis
- Length of time since the burn was sustained
- Prior administration of antibiotics
- The likely pathogens encountered in the burn un (Pathogens vary somewhat from center to center b useful information can be obtained from surveillanc cultures)
- It is important to remember that in burn patients th pharmacokinetics of many antibiotics are often altere and some patients may experience periods during whic sub-therapeutic concentrations extend beyond the pos antibiotic effect unless the dosing regimens are altere appropriately.[60]

Local evidence of invasive wound infection includes:

- Black or brown patches of wound discoloration
- Rapid eschar separation
- Conversion of wounds to full-thickness
- Spreading peri-wound erythema
- Punctuate hemorrhagic sub-eschar lesions
- Violaceous or black lesions in unburned tissue (ecthym gangrenosum)

Initial empirical therapy can be modified subsequentl on the basis of the culture and sensitivity report. Prolonge stay of these patients in the burn unit often leads t colonization by nosocomial resident microflora which tend to be multidrug resistant. It is unrealistic to expect to kee a burn wound sterile. Bacterial counts of less than 10 organisms/gm are not usually invasive and allow skin gra survival rates of >90%. Antimicrobial therapy is directe

Table 9.3: Empirical therapy as directed by the British Society of Chemotherapy, 2006

No. of days after burns	*Treatment history*	*Organism*	*Antibiotic likely advocated*
<or= 5	none	Staph. Or Strept.	Penicillin & Flucloxacillin
<or= 5	yes	-do-	Cefuroxime
6-9	none	Staph or Strept Coliforms	Cefuroxime
6-9	yes	Staph or Strept Coliforms	Flucloxacillin + Gentamycin
> or =10		Pseudomonas	Flucloxacillin + Ceftazidime

Replace beta lactam antibiotic if allergy with clindamycin or macrolide antibiotic by bacterial surveillance through routine tri-weekly sputum, urine, and wound cultures. In addition, punch biopsies for each 18% BSA of burn are obtained for wound monitoring until the wound is closed. Organism identification and *in vitro* sensitivities to antibiotics should be available in 48 hours. Agar diffusion assays can also be performed to test susceptibilities to topical antimicrobials. If quantitative biopsies reveal microbial density of $>10^3$ organisms/gm, a change in topical therapy is indicated. If bacterial counts are 10^5, worsening wound infection should be suspected and rapid histological analysis and Gram stain should be performed to confirm this. At this point, systemic antibiotics must be instituted, and the wound excised.

Antibiotics for Deep Infections

Appropriate systemic antibiotics are administered as indicated. The appropriate use of antibiotics should be based on the following definitions:

Prophylaxis—A preventative or precautionary measure designed to preserve health and prevent the spread of disease.

Perioperative—This may also be considered prophylaxis. It is the administration of systemic antibiotics as a protective measure for any type of surgical intervention. The time frame for administration should be short-lived and is limited to 1 to 3 doses, depending on the operative procedure.

Therapeutic—This is the administration of antibiotics for the treatment of infection. Depending on the infection, therapy may continue for several days.

Systemic antibiotic treatment for burn wound sepsis is continued for at least 72 hours after evidence of sepsis has resolved. If the wounds appear clean, other sources such as the lungs, kidneys and peripheral veins should be suspected as the primary source of infection. The commonly used agents in several situations are Vancomycin, Imipenem, Timentin or Ceftazidime for coverage against the usual strains of *Staph. aureus* and *Pseudomonas aeruginosa* prevalent in the burns unit. Routine perioperative antibiotics should also take ward-endogenous organisms into account. Postoperative antibiotics are continued until quantitative excisional wound biopsies from surgery are identified.

Notes[61, 62]

1. For patients with burns of ≥ 2 weeks' duration and/or who are colonized with multidrug resistant gram-negative bacilli, seek advice from a clinical microbiologist.
2. Modify therapy, if appropriate, in the light of culture and susceptibility test results (blood culture and burn would surveillance cultures).
3. Duration of treatment: 5 days, if a significant pathogen is isolated from blood cultures or the burn wound; 3 days, if a pathogen is not isolated from blood cultures or the burn wound. As there is no or only limited margin for error in patients who are severely ill with suspected infection within five days of sustaining a burn, broad-spectrum cover, with an agent such as cefuroxime, should be administered if an aerobic Gram-negative bacillus cannot be excluded.
4. It is important to remember that in burn patients, the pharmacokinetics of many antibiotics are often altered and some patients may experience periods during which sub-therapeutic concentrations extend beyond the post-antibiotic effect unless the dosing regimens are altered appropriately.
5. Tetanus immunization is important (neonates, the elderly or the debilitated may require immunoglobulin).
6. In case of multiple drug resistant organisms (MDRO), immediate intimation should be given to the Infection control personnel and advice from them may be gathered. MDRO include Methicillin resistant *Staphylococcus aureus* (MRSA), Vancomycin resistant *Enterococcus* (VRE), Extended Spectrum beta lactamase (ESBL) producing Gram-negative bacteria (GNB), MDR. *Pseudomonas* and MDR *Acinetobacter* and recently pan Drug resistant *Pseudomonas* and *Acinetobacter* which are resistant to almost all the known antibiotics.
7. *Staph. aureus* tends to remain localized to the wound, if patient becomes toxic consider Toxic Shock Syndrome (TSS).
8. *Candida* spp. remain localized but rarely do invade. Always prove invasion with this pathogen.
9. Pneumonia is the most prevalent of complications with high mortality. *Staph. aureus* is most commonly implicated. Septic thrombophlebitis is also frequently seen.
10. Daptomycin (4 mg/kg IV q24h) can be used instead of Vancomycin.

Antibiotics for burn wound sepsis where MDRO are seen.[60,61]

- Vancomycin (1gm IV q12h) + Amikacin (10 mg/kg loading then 7.5 mg/kg IV q12h) + Piperacillin 4 gm IV q 4 h) (give ½ q 24 h Piperacillin dose in subeshcar tissue and surgical removal of eschar in 12 hours).

- If resistant bacteria, Piperacillin + Tazobactam may be used instead of Piperacillin.
- If ESBL producing GNB then Imipenem or Meropenem can replace Piperacillin—Tazobactam combination).

Other suggested regimens[63]

- *Pseudomonas* spp.—Tobramycin / Amikacin or Aztreonam + Piperacillin/Ceftazidime/Cefoperazone or Cefepime or Quinolones
- Resistant GNB—Gentamicin or Aztreonam or Amikacin + Piperacillin or Mezlocillin or Third generation Cephalosporin or Cefepime. Quinolones or Trimethoprim-Sulphamethoxazole combination may be kept as reserve in resistant but non ESBL GNB infections
- In ESBL or AMP C producing GNB infections-Metallobeta lactams—Imipenem, Meropenem MUST BE USED. Other group of antimicrobials may be combined if proven as sensitive and no deleterious effect of combination is seen.

Suggested Groups of Antibiotics **to be tested/applied in the Microbiology Laboratory** for Nosocomial isolates as Primary, Primary but selective use and Supplemental or reserved use for each major Group of bacteria[64]

I. Enterobacteriaceae (*Klebsiella* spp, *E.coli*, *Proteus* spp etc.)
   - Primary—Ampicillin, Cefazolin, Caphalothin, Gentamicin, Tobramycin.
   - Primary but selective use—Cefuroxime, Cefepime, Cefotaxime, ceftriaxone, Cefoxitin, Piperacillin, Amoxy-clav, Ampi-sulbactam, Piperacillin-Tazobactam, Ticarcillin-clavulanate, Amikacin, Ciprofloxacin, Meropenem, Imipenem.
   - Reserve—Aztreonam, Ceftazidime, Chloramphenicol, Tetracycline, Newer agents and agents under trial.

II. *Pseudomonas* spp
   - Primary—Ceftazidime, Gentamicin, Tobramycin, Piperacillin.
   - Primary but selective use—Amikacin, Aztreonam, Cefepime, Piperacillin—Tazobactam, Ticarcillin, Imipenem, Meropenem, Ciprofloxacin.

III. *Staphylococcus* spp.
   - Primary—Oxacillin (group representative of beta lactams drugs and resistance to this eliminates the whole group including even metallobetalactams such as imipenem and meropenem), Penicillin, Erythromycin/Azithromycin/Clarithromycin, Trimethoprim—Sulphamethoxazole.
   - Primary but selective use—Vancomycin, Teicoplanin, Daptomycin, Linezolid, Doxycyclin/Tetracyclin, Rifampicin(rifampicin should bever be used alone in therapy).
   - Reserve—Ciprofloxacin or Ofloxacin or Levofloxacin, Moxifloxacin, Synercid (Quinupristin and dalfopristin combination), Gentamicin.

IV. *Enterococcus* spp.
   - Primary—Ampicillin, Penicillin.
   - Primary but selective use—Vancomycin, Daptomycin, Linezolid, Synercid.
   - Reserve—High level Gentamicin and Streptomycin.

V. *Acinetobacter* spp.
   - Primary—Ceftazidime, Ampicillin-sulbactam, Imipenem, Meropenem, Gentamicin, Tobramycin.
   - Primary but selective use—Cefepime Cefotaxime, Ceftriaxone, Piperacillin, Piperacillin-tazobactam, Ticarcillin-clavulanate, trimethoprim-sulphamethoxazole.
   - Reserve—Colistin, Polymyxin B (to be used in highly resistant MDR *Acinetobacter* and *Pseudomonas* spp).

VI. *Burkholderia* spp.
   - Primary—Trimethoprim-sulphamethoxazole
   - Primary but selective use—Ceftazidime, Minocycline, Meropenem.

VII. *Stenotrophomonas* spp.
   - Primary—Trimethoprim-sulphamethoxazole
   - Primary but selective use—Levofloxacin Minocycline.

The oral antibiotics (usually tested in the primary categor for each group of bacteria) included in the above list ar more for mild burn wounds and infections which do no warrant parenteral antibiotics. This in the case of bur wounds can be omitted altogether and topical agents can b used instead of oral agents, the choice of which lies in th clinician's experience.

The selective use category of antibiotics is primaril parenteral and is advocated for serious infections such a the burn wound septicemias and other deep infections relate to burn wounds (Table 9.4). For polymicrobial infection and infections in multiple sites also, a combination o antimicrobials is advocated as has been suggested previousl in this chapter. Reserve drugs are used in special situation where highly drug resistant pathogens are expected.

A brief mention of some of the newer agents availabl for use, in the various sub-categories of bacteria observe in the nosocomial setting is enlisted beneath. However, thi

Table 9.4: New antibiotics and older antibiotics with new indications or treatment options [65]

Antibiotic	*Drug class*	*Initial development period*	*Antibacterial spectrum*	*Limitations/adverse effects or new issues*	*Current status*
New agents Gemifloxacin, garenoxacin, sitafloxacin	Fluoroquinolone	Early 1960s	Variety of gram-positives (not VRE, MRSA) and gram-negatives	Resistance already established in some places, due to widespread ciprofloxacin use. Role uncertain, given availability of older agents	Garenoxacin development halted
Ramoplanin	Glycolipodepsi-peptide	Early 1980s	Gram-positives, including VRE	Not absorbed orally — used for temporary suppression of faecal VRE colonisation	Not US FDA-approved. In Phase III trials
Daptomycin	Lipopeptide	Late 1980s	Gram-positives: VRE, MRSA, SA-RVS	Myalgia at high doses	US FDA-approved
Linezolid	Oxazolidinone	Late 1980s	Gram-positives: VRE, MRSA, SA-RVS, some *Mycobacteria* spp., *Nocardia* spp.	Haematological toxicity, especially thrombocytopenia; peripheral neuropathy (rare)	US FDA approved
Quinupristin, dalfopristin	Streptogramin	Early 1990s	Gram-positives: VRE (not *Enterococcus faecalis*), MRSA, SA-RVS	Infusion site pain, arthralgia, myalgia	US FDA approved
Oritavancin, dalbavancin	Novel glycopeptides	Late 1990s	Gram-positives: VRE (oritavancin only), MRSA, SA-RVS	Toxicity uncertain	Not US FDA-approved. In Phase III trials
Telithromycin, cethromycin	Ketolide (related to macrolides)	Late 1990s	Typical and atypical respiratory pathogens, including *Mycoplasma*, *Chlamydia* spp.	Hepatotoxicity, blurred vision, QT changes on ECG	US FDA approed
Tigecycline	Glycylcycline (semisynthetic derivative of minocycline)	Late 1990s	Gram-positives: VRE, MRSA, SA-RVS; some gram-negatives	Nausea, vomiting, headaches	US FDA approved

Contd.

Contd.

Antibiotic	*Drug class*	*Initial development period*	*Antibacterial spectrum*	*Limitations/adverse effects or new issues*	*Current status*
Older agents					
Penicillin G	β-Lactam	Late 1930s	Gram-positives: streptococci, enterococci, *Clostridium*, *Listeria* spp.	Continuous infusion provides high serum levels for intermediate-resistant strains (e.g., some VRE). Suitable for hospital-in-the-home treatment in some cases	
Colistin, polymyxin B	Cationic peptides	1950s	Gram-negatives, including *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Klebsiella pneumoniae*	Renal toxicity, neurotoxicity	
Amphotericin B	Polyene	Late 1950s	Most fungi	Preliminary data suggest that continuous infusion may reduce renal toxicity and hence reduce the need to switch to newer, more expensive liposomal analogues	
Gentamicin	Aminoglycoside	Early 1960s	Many gram-negatives	Meta-analyses suggest a once-daily dose of 4–6 mg/kg daily † is associated with reduced renal and ototoxicity, but efficacy is similar to multidose regimens	

ECG = electrocardiogram. FDA = Food and Drug Administration. MRSA = methicillin-resistant *Staphylococcus aureus*. SA-RVS = *Staphylococcus aureus* with reduced vancomycin susceptibility. VRE = vancomycin-resistant enterococci.† Recommended dose for an adult with normal renal function.

is an ever expanding field and even at the time of publication of this document several more agents will have been tried and tested for human use.

Multidrug resistant bacteria for which there are currently few available treatment options.

Gram-negatives

- *Acinetobacter baumannii*
- *Pseudomonas aeruginosa*
- *Stenotrophomonas maltophilia*
- ESBL and very rare MBL *Klebsiella pneumoniae*

Gram-positives

- Methicillin-resistant *Staphylococcus aureus*
- Vancomycin intermediate or resistant *Staph.aureus*
- *S. aureus* with reduced susceptibility to vancomycin and/or linezolid
- Methicillin-resistant coagulase-negative staphylococci
- Vancomycin-resistant enterococci.

FUNGAL INFECTIONS

Invasive burn wound infections due to *Candida* spp., *Aspergillus* spp., and other opportunistic fungi (including *Alternaria* spp. *Fusarium* spp., *Rhizopus* spp. and *Mucor* spp.) are important emerging causes of late-onset morbidity and mortality in patients with major burns and severely perturbed immune systems.[66] Thermally injured children are at particular risk for fungal infections involving the burn wound. Such infections usually occur after the second week of thermal injury, usually following a period of burn wound colonization with fungi found in the surrounding environment and/or, in the case of *Candida* spp., from the patient's own gastrointestinal or upper respiratory tract flora.[67]

Infections are most commonly due to *Candida* spp. and *Aspergillus* spp. and generally appear as a darkening of the burn wound area, with an appearance similar to that of ecthyma gangrenosum.[68] Burn wound infections due to zygomycetes such as *Rhizopus* spp. and *Mucor* spp., although very rare, are important because of their remarkable ability to spread rapidly across fascial tissue planes and to invade the vasculature and are associated with a very high mortality rate. Diagnosis of fungal infections of burn wounds is most reliably made by histological examination of burn wound tissue samples along with culture of the biopsy sample. Aggressive wide surgical excision of the infected areas of the burn wound before the development of deep invasion, combined with systemic antifungal agents, may lead to an improved chance of survival.[68] The use of a laminar-airflow isolation room, in conjunction with optimal local wound care, appears to decrease the incidence of both disseminated and local fungal infection. Topical prophylaxis with an antifungal cream such as clotrimazole or nystatin may lead to a reduced incidence of burn wound infections and septicemia.[67]

If fungal etiology is detected on histopathology, culture samples to detect the infecting genus and species are necessary because the growing armamentarium of antifungal agents have varying activity. Amphotericin B was once the agent of choice, but some facilities have seen increased rates of infections with *Fusarium* species and *Aspergillus terreus,* which are innately resistant to amphotericin B. In these cases, voriconazole is often used. A new agent, posaconazole, may have broader antifungal activity but is limited as it is available only in an oral formulation. In case of proven *Candida* spp invasive disease Amphoteracin B or Ketoconazoles may be initiated. Liposomal Amphoteracin B has wider absorbance and efficacy, fewer side effects but much more expensive. Caspofungin, an echinocandin, is another new antifungal in the horizon which shows dramatic results in therapy of resistant aspergillosis and candidiasis.

VIRAL INFECTIONS

Viruses of the herpes virus group, particularly herpes simplex virus and varicella-zoster virus but less commonly cytomegalovirus are rarely reported but increasingly recognized causes of wound infections in thermally injured patients. Such infections may be due to primary viral infection, reactivation of latent virus, or exogenous reinfection in previously infected patients. Burn wound infections due to herpes simplex virus occur most commonly in healing or recently healed partial-thickness burns particularly those in the nasolabial area, or occasionally from skin graft donor sites, while those due to cytomegalovirus typically involve full-thickness burns. In the case of herpes simplex virus, clusters of small vesicles or vesiculo-pustules may be found within or around the margins of the burn wound, usually occurring anywhere from 2 to 6 weeks following thermal injury. Herpetic viral infections of the burn wound are usually self-limiting, although systemic spread may occur. Diagnosis is most reliably made by histological examination or culture of burn wound biopsy specimens.

COMPLICATIONS

- Burn wound infections are often the source of bacteria responsible for other systemic infections including bloodstream infections and pneumonia.
- This can lead to Systemic Inflammatory Response Syndrome (SIRS) and Multiple Organ Dysfunction Syndrome (MODS) and death.

SUMMARY

Burn care involves a multisystem approach, requiring numerous health care providers to be involved in the process. The treatment of such infections is often prolonged and is associated with an increased risk for complications, including iatrogenic episodes. As such, continued diligence is needed.

Consultation with an infectious disease specialist is suggested if multidrug-resistant bacteria are present. The basal metabolic rate increases as the percentage of TBSA burned increases. Early enteral feeds should be started. Patients may be as active as they can tolerate. Aggressive physical and occupational therapy of extremity injuries is necessary to prevent long-term morbidity.

A primary focus during hospitalization is to prevent nosocomial transmission of multidrug-resistant pathogens, especially in patients with a greater percentage of TBSA burn (approximately 30%) or who are known to be colonized with multidrug-resistant bacteria such as MRSA, vancomycin-resistant *Enterococcus* species, or gram-negative bacteria known to develop resistance (e.g., *Pseudomonas*, *Klebsiella, Acinetobacter* species). Hand hygiene should be aggressively implemented. Standard precautions should be used in the care of all patients with burn injuries. Patients should be managed in single rooms, if possible, with use of contact precautions. Gowns and gloves should be used when contact is expected with infected material or open wounds. Masks and caps should be implemented based on the presence of multidrug-resistant bacteria. The use of individual-patient equipment should be considered. Aggressive infection-control procedures should be undertaken when transferring patients between facilities because of the risk of transferring multidrug-resistant bacteria. The prevention of burn wound infection is a team approach that includes the support of surgeons, nurses, infection-control providers and infectious disease physicians. Emphasis on early wound care, infection-control practices and long-term rehabilitative care is necessary to improve the morbidity and mortality associated with burns. Early removal of full-thickness burned tissue, as well as early definitive wound closure and strict enforcement of infection-control procedures, is necessary to mitigate p outcomes

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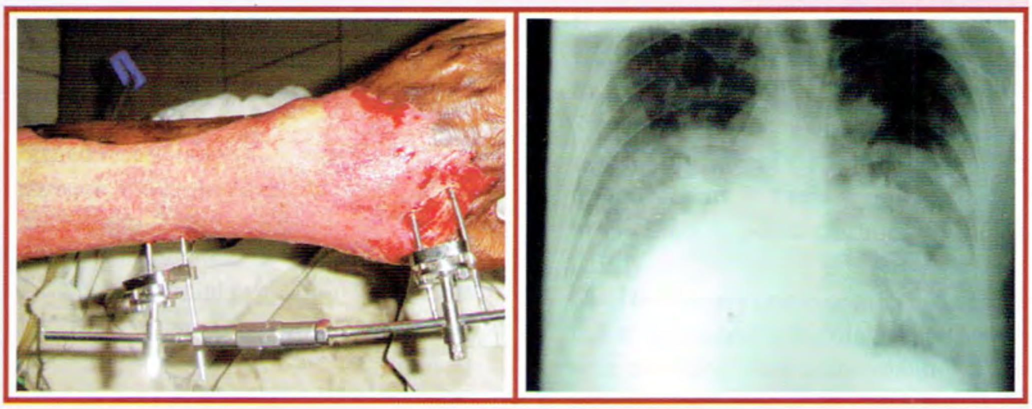
2
Section

Early Care in Burns



Chapter 10

Initial Evaluation and Resuscitation of Burn Patient

Madhuri A Gore

INTRODUCTION

The goal of burn management is to achieve wound closure at the earliest. The efforts in this direction must begin at the first interaction between the victim and the health care worker. The decision regarding the need for admission to burn unit or management on outpatient basis should be protocol based to minimize errors.

The plan of management of patients with large burns that require inpatient care is usually determined by the severity of the alterations in physiology due to the burn injury. These patients require a coordinated approach that involves a specialized team. Hospitalization is divided into 4 general phases:

1. Initial evaluation and resuscitation
2. Initial wound excision and biologic closure
3. Definitive wound closure
4. Rehabilitation and reconstruction.

An orderly and systematic initial evaluation is valuable for delivery of comprehensive care. The early management extends from first aid at site to complete resuscitation of the patient and remains the chief determinant of final outcome especially in a patient with major burn.

FIRST AID: THERMAL BURNS

First aid to a burn patient is generally provided by the relatives, friends and other persons present at the site of burn accident. A specialist burn surgeon is not present at the site. However, the role of a burn specialist in carrying out first aid in burns is to educate the masses about the basics of burn injuries and do's and don'ts. Media has a great role to play in dissemination of this knowledge. This information should also be given to school children in their curriculum. All police and fire brigade personnel, etc. should receive this training as part of their induction into service as they may be required to provide first aid to victims of burn disasters. Some of the points mentioned below may be possible only in the nearest medical facility.

Management on Site

First make the scene safe for the rescuer.

1. **If an individual is actually in flames, instruct the victim to STOP – DROP – and ROLL. Douse the fire with water or sand.**
2. Switch off the mains in the event of electric burns.
3. **Check for signs of circulation, breathing, coughing or movement.** If there is no breathing or other sign of circulation, begin cardiopulmonary resuscitation (CPR).
4. **Cool the burn** - Immerse affected extremity in bucket full of tap water till burning sensation disappears OR hold the burnt part under running tap water till the burning pain disappears OR apply cold compresses on the burnt area.
5. **Do not use ice** as this can cause cold injury and further damage to skin.
6. **Do not immerse severe large burns in cold water.** Doing so can lead to hypothermia.
7. **Do not** remove clothes that are adherent to wounds.
8. **Do not** apply any local agent on the wound (especially colorful agents like gentian violet, mercurochrome) as this interferes with the evaluation of burn depth and extent.

9. *Do not break the blisters. Broken blisters are more* vulnerable to infection.
10. **Cover the area of the burn, this reduces the pain.** Use a sterile bandage or clean cloth. Sterile polyethylene drape is very useful for covering the burnt area as it is non-adherent and helps to avoid hypothermia. Do not use fluffy cotton wool–it irritates the burn wound.
11. If the patient is conscious, give salt – sugar solution to the patient orally.
12. Shift patient to appropriate facility for management (Table 10.1).

Table 10.1: Key points in First Aid

1. Stop the burning process
2. Maintain airway.
3. If a burnt patient is unconscious, think of smoke inhalation and/ or of associated head injury.
4. If a burnt patient is breathless, think of inhalation injury, chest trauma and / or aspiration.
5. If a burnt patient is in shock early after the incident, think of associated chest or abdominal trauma causing blood loss or of spinal trauma.
6. Shift the patient to appropriate burn care facility

FIRST AID: CHEMICAL BURNS

Chemical burn injuries are not hyperthermic, but are due to tissue reactions to noxious substances, including oxidizing agents, reducing agents, corrosives, protoplasmic poisons, desiccants and vesicants (Fig. 10.1).

1. **Remove the cause of the burn** by flushing the chemicals off the skin surface with cool, running water for 15 minutes or more. Clothes soaked in chemical should be removed. Deep alkali burns should be irrigated for 24 hours. A balanced saline solution is preferable for irrigation of mucosal surfaces or eyes. Dilution of the chemical is of paramount importance and not neutralization. Neutralization can produce exothermic damage as well. If the burning chemical is a powder-like substance such as lime, brush it off the skin before flushing.

   Initial copious hydrotherapy is indicated for all chemical burns except those caused by dry-lime, phenol, concentrated sulphuric acid, sodium metal and muriatic acid; they are either not miscible with water or react with water exothermically.
2. **Antidotes** are helpful for burns from hydrofluoric acid, phenol and white phosphorous.[1]

   Hydrofluoric acid (HFA) causes liquefaction necrosis of the subcutaneous tissue and can penetrate to bone.

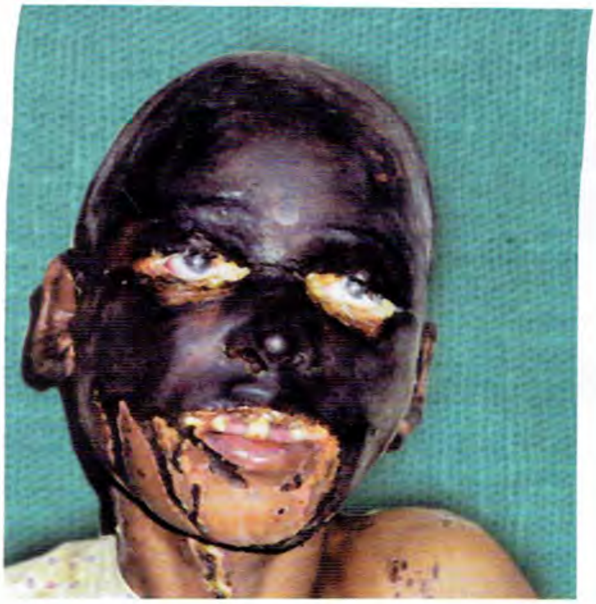


Fig. 10.1: Chemical burns of face involving the eyes

   Systemic complications of HFA toxicity include hypocalcemia and pulmonary edema. HFA wounds should be covered with a 10% calcium gluconate solution mixed to a slurry with a water soluble ointment or infiltrated if the burn is excruciatingly painful.

   Phenol is an acidic alcohol which produces local coagulation and systemic toxicity in large doses, including fatal arrhythmias. Acute phenol burns are treated topically by irrigation with polyethylene glycol solution.

   White phosphorus is contained in grenades and anti-personnel mines. Once particles are embedded in skin, white phosphorus causes burn by both chemical and thermal reactions, as particles are spontaneously ignited with prolonged exposure to air. Patients should be submersed in water until embedded particles can be surgically debrided. The skin is washed with a solution of 5% sodium bicarbonate, 3% copper sulfate and 1% hydroxyl-cellulose to blacken the particles and aid earlier identification. Prolonged exposure to copper sulphate solution can induce coagulopathies.
3. **Remove clothing or jewellery** that has been contaminated by the chemical.
4. **Wrap the burned area loosely** with a dry, sterile dressing or a clean cloth.
5. **Patient with even a minor chemical burn on** the eye, hands, feet, face, groin or buttocks or over a major joint should be treated at a burn center.

FIRST AID: ELECTRICAL BURNS

High-voltage electrical injuries are classified as major burns due to associated massive tissue damage. On average, high-voltage cutaneous involvement is only 10-15% BSA, but this visible injury is only a small portion of the overall tissue destruction (Fig. 10.2). The human body serves as a volume conductor to electricity. Current flow is, therefore, concentrated in the extremities by their narrowing. Since bone has the highest resistance to electrical current, conduction through bone produces the greatest amount of heat. For this reason, damage to muscle by electricity is greatest at tendinous attachments and periosteal regions. Massive muscle destruction causes myoglobinemia. Precipitation of myoglobin in the renal tubules can produce acute tubular necrosis and acute renal failure. In addition, high-voltage electrical burns are commonly associated with dislocations, fractures, vertebral injuries, myocardial damage, neurological sequel and intra-abdominal injuries due to concomitant trauma. The most common cause of early mortality is cardiopulmonary arrest due to induced fibrillation.

While helping someone with an electrical burn and waiting for medical help, following steps are recommended:

1. **Look first. Do not touch.** The person may still be in contact with the electrical source. Touching the person may allow the current to pass through the rescuer.
2. **Turn off the source of electricity if possible.** If this is not possible, move the source away from the injured person using a non-conducting object made of cardboard, plastic or wood.
3. **Check for signs of circulation breathing, coughing or movement.** If absent, begin cardiopulmonary resuscitation (CPR) immediately.
4. **Prevent shock.** Lay the person down with the head slightly lower than the trunk and the legs elevated.
5. **Cover the affected areas.** If the person is breathing, cover any burned areas with a sterile gauze bandage, if available, or a clean cloth. Do not use a blanket or towel. Loose fibers can stick to the burns.

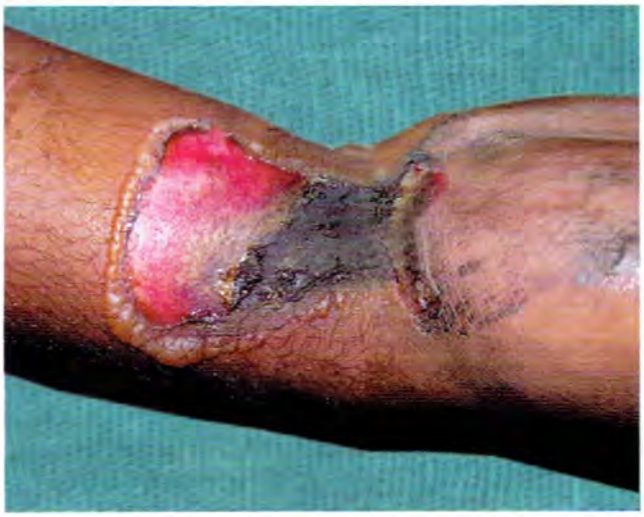


Fig. 10.2: Electrical burns involving wrist – Even though external injury appears to be a minor burn but the underlying tissue destruction is extensive involving muscles, nerves, vessels and bone

TRANSPORTATION

Almost all burn patients in India are transported, at least initially, by the relatives of the victim to the nearest medical facility. Only an extremely small number of them have inhalational injury significant enough so as to require immediate control of airway and intubation or oxygen therapy to avoid complications and death. The burn patients, as opposed to the multiple trauma patients with active blood loss, are unlikely to develop shock or airway obstruction in the first one hour or so post burn. Commonly, it is only during disasters with a number of casualties occurring together that the ambulances are rushed to the site and/or the police/fire brigade personnel transport the victims. In such situations only, there is a possibility of intravenous fluid being started or oxygen being administered to patients on site and continued during transportation. In the large cities where specialized burn centers are available, the patient may be transported directly to it. Otherwise, they are transported to the nearest medical facility. Here, the initial treatment is started and then, transfer to a burn facility is considered.

The procedures that should be performed in the pre-hospital setting (on site) by trained paramedics before and during transportation to the initial medical facility or from the latter by medical personnel before a long transportation to a specialised burn care center are:

1. **Stop the burning process:** Use water for smouldering clothes, chemicals, hot tar, etc.
2. **Check airway:** If deep facial burns are present and/or there is a history of smoke exposure with obtundation or stridor or other symptoms of impaired airway – **INTUBATE**
3. **Initiate high flow oxygen therapy** if history of smoke exposure is present, to treat carbon monoxide toxicity.
4. **Check for other Injuries** especially to chest (rib fractures, pneumothorax, etc.) (Fig. 10.3) due to other trauma suffered in the accident.
5. Assess hemodynamic stability and perfusion by looking for evidence of impending burn shock.

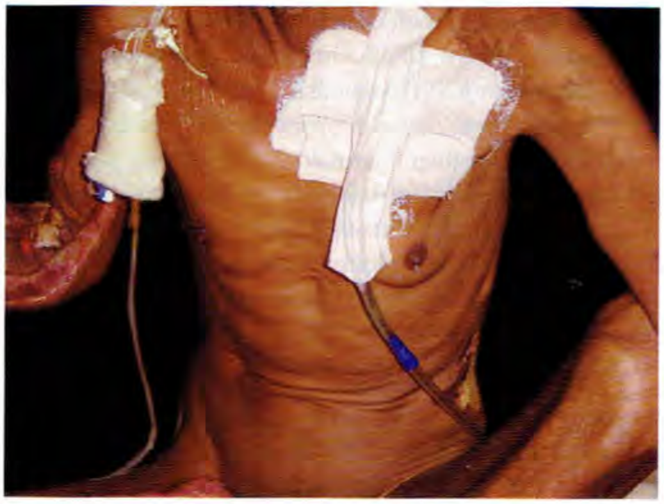


Fig. 10.3: Chest tube drainage in situ for pneumothorax because of fracture ribs following fall from height due to electric injury

6. **Start I / V infusion** if burn size is greater than 15% of total body surface (or if there are other injuries) infuse lactated Ringers

   An estimate of fluid requirement is:

   a. 15-25% BSA = 500 ml per hour
   b. 25-50% BSA = 750 ml per hour
   c. > 50% BSA = 1 Liter per hour

   The transport vehicle should have adequate supply of crystalloids as well as colloids needed for resuscitation.
7. **Assess depth and size of burn while** keeping the patient warm (remove wet clothes). Cover patient with clean and dry dressing to conserve heat.
8. **High tension (> 1000 Volts) injury**—Look for cardiac abnormalities, entrance and exit sites and other injuries.
9. **Remove any constricting objects** (Rings, Bangles, etc)
10. **Information to receiving center should include:**

    a. Neurologic status at scene
    b. Smoke exposure and airway status
    c. Hemodynamic stability
    d. Approximate size, depth and areas involved in burn

For transfer from the initial medical facility to a burn unit or higher center, the ambulance used for transporting the victim should have all the resuscitation equipment and a competent team of qualified doctor and nurse/paramedic. Safety during transfer is more important than speed. The transport should be by the fastest mode available. It must be remembered that initial treatment of an extensively burnt patient has to do more with care of airway, breathing and circulation and resuscitation with intravenous fluids which comes in the domain of every qualified medical practitioner than the wound itself which has to be simply protected from further contamination. This much treatment is presently available in almost all parts of the country. Hence, the practice of transporting the patient in great hurry to a higher center without providing simple care should be discouraged (Table 10.2).

Table 10.2: Key points during transportation

- Assess airway. Intubate if necessary.
- Secure two large bore intravenous lines for > 20% burn extent.
- Start intravenous Ringer's Lactate infusion at desired rate.
- Insert nasogastric tube.
- Introduce per-urethral catheter to monitor urine output during transport.
- Apply topical agent. Wrap in sterile clean drape. Avoid hypothermia.
- Carry resuscitation equipment and develop competence to use it.
- Intimate receiving center.
- Provide information about patient's condition, treatment given and actual incident.

ADMISSION CRITERIA

Burn Surface Area Assessment

An accurate estimate of the size of the burn is important for treatment and transfer decisions. The size or extent of the burn can be estimated in a number of ways. When calculating burn area, erythema should not be included. This may take a few hours to fade, so some overestimation is inevitable if the burn is estimated acutely.

Wallace's 'Rule of 9' is a quick and practical way of estimating the surface area affected by burn. The adult body configuration is divided into anatomic regions that represent 9% or multiples of 9% of the total body surface area (Fig. 10.4).

- Head and neck, 9%
- Front of trunk, 18%
- Back of trunk, 18%
- Upper limb each, 9%
- Lower limb each, 18%
- Perineum and external genitalia,1%

Body surface area proportions differ considerably for children. Using 'Rule of 9' for a child can lead to wrong estimation of the extent and hence improper fluid resuscitation. For patients younger than 15 years, a more precise method of burn size estimation must be used.[2]

Lund and Browder's chart: Perhaps, the most accurate method is the age-specific Lund-Browder's chart (Table 10.3) that compensates for the changes in body proportions

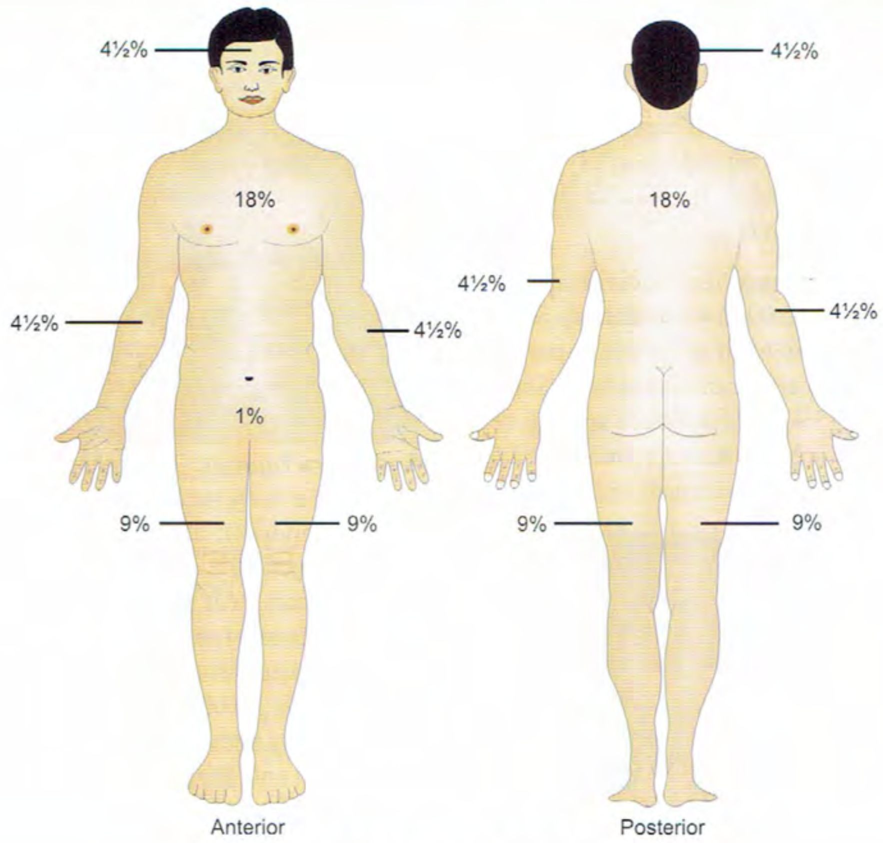
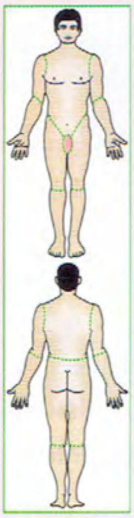


Fig. 10.4: Wallace's Rule of 9

Table 10.3: Lund and Browder's chart for estimating area of burns

Area	*Birth to1 year*	*1 to 4 years*	*5 to 9 years*	*10 to 14 years*	*15 years*	*Adult*	*2^{nd}**	*3^{rd}**	*TBSA*
Head	19	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Anterior trunk	13	13	13	13	13	13			
Posterior trunk	13	13	13	13	13	13			
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5			
Genitalia	1	1	1	1	1	1			
Right upper arm	4	4	4	4	4	4			
Left upper arm	4	4	4	4	4	4			
Right lower arm	3	3	3	3	3	3			
Left lower arm	3	3	3	3	3	3			
Right hand	2.5	2.5	2.5	2.5	2.5	2.5			
Left hand	2.5	2.5	2.5	2.5	2.5	2.5			
Right thigh	5.5	6.5	8	8.5	9	9.5			
Left thigh	5.5	6.5	8	8.5	9	9.5			
Right leg	5	5	5.5	6	6.5	7			
Left leg	5	5	5.5	6	6.5	7			
Right foot	3.5	3.5	3.5	3.5	3.5	3.5			
Left foot	3.5	3.5	3.5	3.5	3.5	3.5			
Total:	100	100	100	100	100	100			

The figures can be colored in red for full-thickness burns (3rd*) and blue for partial-thickness burns (2rd*). TBSA – Total body surface area.

with growth. The burn is drawn on a cartoon figure and an associated age-specific table is used to calculate the body surface area involved. This provides more accurate distribution of body surface area and is especially valuable for burnt children. For a burn unit, adoption of this burn extent estimation chart is advisable. Its use also facilitates comparison of data from different units.

Palmar surface: *The* surface area of an individual's palm (including fingers) is roughly 0.8% of total body surface area [3], though it is taken as 1% for all practical purposes (Fig. 10.5). Palmar surface area can be used to estimate relatively small burns (< 15% of total surface area) or very large burns (> 85%, when unburnt skin is counted) or areas of irregular or non-confluent burns.

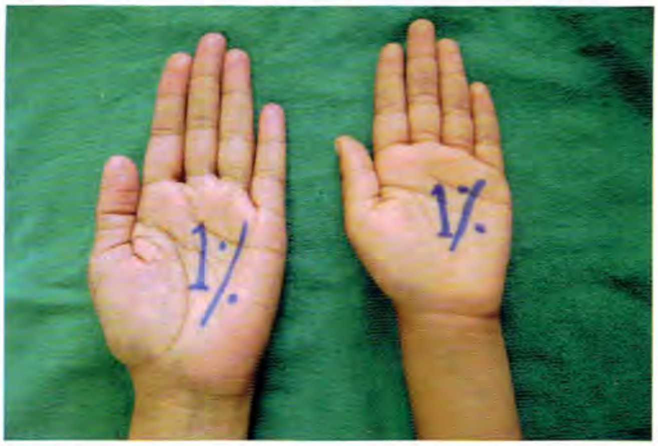


Fig. 10.5: Palmar surface of hand with fingers closed representing 1% of body surface area in adult (Left) and child (Right)

While assessing the extent of burn, it is important that the entire body surface is exposed and assessed. The environment should be kept warm and hypothermia should be avoided. Burns of the pigmented skin can be difficult to assess and it may be necessary to remove all the loose epidermal layers to unveil the exact burn size.

Burn Unit Referral

To standardize the treatment categorization, American Burn Association has identified three treatment categories for burn patients: major, moderate and minor burn injuries[4] (Table 10.4).

Major Burn Injuries: This group includes partial thickness burns with a body surface area greater than 25% in adults (20% in children); all full thickness burns with a body surface area of 10% or greater; all burns involving hands, face, eyes, ears, feet and perineum; all inhalation injuries; electrical burns of any extent; complicated burn injuries involving fractures or other major trauma; and all poor risk patients (Fig. 10.6). Major burn patients should be transported to a burn center. The choice of a hospital depends on distance and time, the patient's burn complications (respiratory condition, shock) and availability of bed. The American Burn Association emphasizes the importance of direct communication and transfer agreements among hospitals. This system is highly desirable though non-existent in India at present. If the seriousness of the patient's injury dictates transportation to the closest emergency department of a hospital, then subsequent transfer to a burn

Table 10.4: American Burn Association's grading system for burn severity and disposition of patients

	Type of burn		
	Minor	*Moderate*	*Major*
Criteria	<10 percent TBSA burn in adult <5 percent TBSA burn in young or old <2 percent full-thickness burn	10 to 20 percent TBSA burn in adult 5 to 10 percent TBSA burn in young or old 2 to 5 percent full-thickness burn Excluding High-voltage injury Suspected inhalation injury Circumferential burn Concomitant medical problem predisposing the patient to infection (e.g., diabetes, sickle cell disease)	>20 percent TBSA burn in adult >10 percent TBSA burn in young or old >5 percent full-thickness burn High-voltage burn Known inhalation injury Any significant burn to face, eyes, ears, genitalia or joints Significant associated injuries (e.g., fracture, other major trauma)
Disposition	Outpatient management	Hospital admission	Referral to burn center

Young = patient younger than 10 years of age; adult = patient 10 to 50 years of age; old = patient older than 50 years of age. *Adapted from*: Hospital and prehospital resources for optimal care of patients with burn injury: guidelines for development and operation of burn centers. American Burn Association. J Burn Care Rehabil 1990; 11:98-104, with additional information from Hartford CE. Care of outpatient burns. In: Herndon DN, ed. Total burn care. Philadelphia: Saunders, 1996:71-80

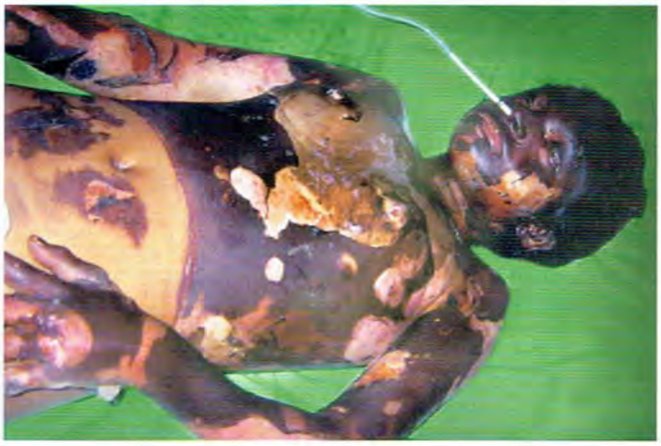


Fig. 10.6: Major burn injury

center should be arranged after cardiopulmonary stabilization and intravenous fluid therapy for shock.

Moderate, Uncomplicated Burn Injuries: This second group includes partial thickness burns with a body surface area of 15 to 25% in adults (10 to 20% in children), full thickness burns with a body surface area of less than 10% and burns that do not involve eyes, ears, face, hands, feet or perineum. Excluded from the group are electrical injuries, complicated injuries (fractures), inhalation injuries and all poor risk patients (elderly patients and patients with an intercurrent disease). Most of the patients in this group should receive emergency care at the site of the injury and be transported directly either to a hospital where burn care expertise is available or to a hospital with a burn center.

Minor Burn Injuries: The third group includes partial thickness burns with a body surface area of less than 15% in adults (10% in children), full thickness burns with a body surface area of less than 2% and burns not involving eyes, ears, face, hands, feet or perineum. It excludes electrical injuries, inhalation injuries, fractures and all poor risk patients. The patients in this group may be transported to a hospital emergency department where definitive care begins.

In Indian setting, it is highly desirable to provide treatment to patients with moderate and minor burns in a Level 2 hospital with support from burn unit at Level 1(tertiary care) hospital whenever necessary. This can control the patient load in the few burn centers available and hence help improve the quality of care.

Burn Unit Admission Criteria

A burn unit may treat adults or children or both.

Burn injuries that should be treated at a burn unit include the following:

1. Partial thickness burns greater than 10% total body surface area (TBSA)
2. Burns that involve the face, hands, feet, genitalia, perineum or major joints
3. Full thickness burns in any age group
4. Electrical burns, including lightning injury
5. Chemical burns
6. Inhalation injury
7. Burn injury in patients with pre-existing medical disorders that may complicate management, prolong recovery or affect mortality
8. Any patient with burns and concomitant trauma (such as fractures) (Fig. 10.7) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit. Physician judgment is necessary in such situations and should be in concert with the triage protocols.
9. Burned children in hospitals without qualified personnel or equipment for the care of children.
10. Burn injury in patients who require special social, emotional or long-term rehabilitative intervention.

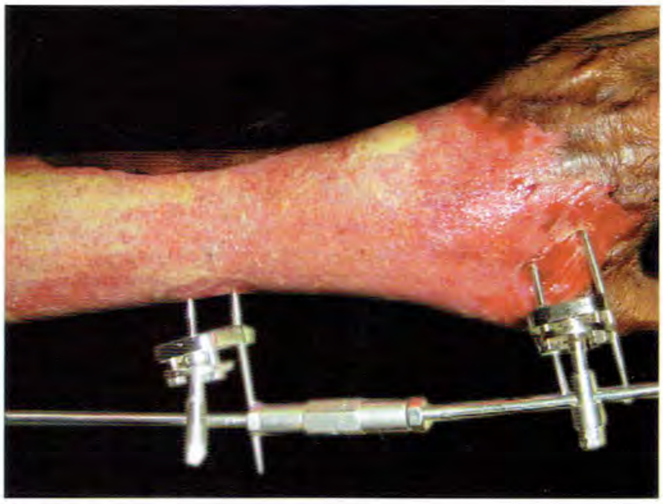


Fig. 10.7: Fracture of lower end of radius, along with burns in the forearm and hand, due to fall, after sustaining electric burns

Selection for outpatient care: Outpatient management is best coordinated with the burn unit's team of doctors, nurses and therapists because their expertise may facilitate attaining optimal outpatient results. Several factors are relevant to a decision regarding the outpatient care. No question of airway compromise should exist. The wound must be small enough so that intravenous fluid resuscitation is not required. The patient must be capable of oral intake of an adequate amount of fluids. Generally, serious burns on the face, ears, hands, genitals or feet should be initially managed in an inpatient setting. The patient and his/her family must be able to comply with an outpatient care plan. An adult caregiver must be present with a child being managed in an outpatient setting. A family member must be available to perform necessary wound cleansing, inspection and dressing applications because most patients cannot do these themselves. The family must have adequate transportation to return for follow-up visits and unexpected emergency visits. Specific conditions that mandate an early return should be detailed. Particularly important are pain and anxiety associated with wound care to the degree that wound care is compromised. This can be suspected if the wound shows signs of infection or appears deeper than appreciated at the time of the initial examination. In such a situation the patient should be admitted. If abuse is suspected, outpatient management is contraindicated. If on initial examination surgery is clearly needed for a full-thickness burnt area, the patient should be admitted. Despite all of these stipulations, most patients with small burns can be successfully managed in an outpatient setting.

OUTPATIENT WOUND CARE STRATEGIES [5]

The components of outpatient burn care include:

1. Patient and family teaching,
2. Wound cleansing,
3. Choice of topical agent or membrane dressing,
4. Pain control,
5. Early return instructions,
6. Follow-up clinic visits,
7. Long-term follow-up.

Wound cleansing and dressing techniques must be taught to whosoever will be doing the dressing changes. Documenting this teaching is ideal.

INPATIENT MANAGEMENT

A major burn is defined as a burn covering 25% or more of total body surface area (more than 20 % for children), but any injury over more than 10% should be treated similarl Rapid assessment is vital. The general approach to a majo burn can be extrapolated to managing any burn.

Early Management of Major Burn

The burn wound should never take precedence ove potential life threatening conditions. As described b the American College of Surgeons Committee on Trauma evaluation of the burn patient is organized into a primar survey and a secondary survey using modification of the methodology of Advanced Trauma Life Support Course.[6]

Primary survey: The emphasis is on detection and treatment of life threatening conditions.

A – Airway patency - Heat affects primarily the oral and nasal cavity, supraglottic area of the pharynx and causes edema and mechanical upper airway obstruction. The gas, particle and chemical components of the smoke affect primarily the airway mucosa and cause the actual chemical burn. The initial smoke injury occurs shortly after exposure within 30 minutes to 1 hour, but the ensuing intense inflammatory reaction evolves over a period of hours to days resulting in lung damage. The initial edema usually wears off within a day or two and is evident by air leak by the sides of the endotracheal tube. Inhalation injury should be suspected in all of the following situations (Fig. 10.8):

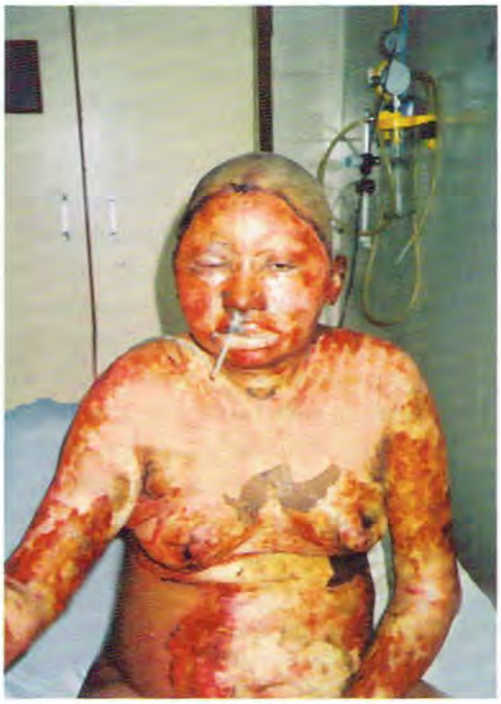


Fig. 10.8: Burn distribution suggestive of inhalation injury

- Individuals who sustained injury in a closed space,
- Patients with extensive burns or with burns of the face,
- Patients who were unconscious at the time of injury,
- Patients with singed nasal and facial hair, hoarseness or wheezing and
- Patients who are coughing up carbonaceous sputum.
- Explosions with burns to head and torso,
- Carboxyhemoglobin level > 10%.

Pulmonary injury is known to result in substantially increased burn mortality and morbidity. When indicated, early intubation and positive pressure ventilation has been shown to improve outcome (Flow chart 10.1).

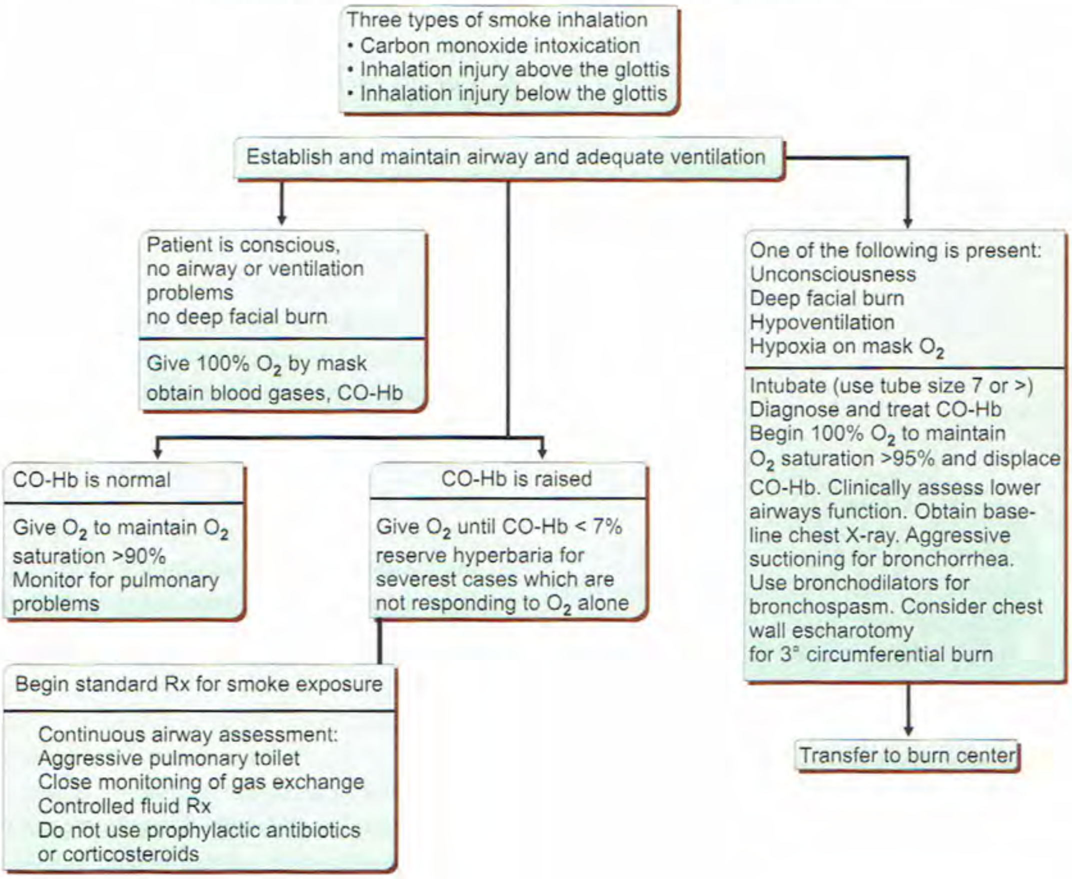
A number of techniques have been used to assess the degree of supraglottic and subglottic injury and to determine the need for endotracheal intubation. Fiberoptic bronchoscopy or laryngoscopy and virtual bronchoscopy can reveal physical evidence of mucosal injury. In the author's experience, in patients with clinical suspicion of inhalation injury, fiberoptic bronchoscopy confirmed the suspicion in 93% of patients (unpublished data). Hence, in the absence of diagnostic modalities, high index of suspicion should guide the management.

As the injury process is progressive during the first 18 to 24 hours, initial appearance does not accurately predict the severity of subsequent airway compromise. With increase in edema, it becomes much more difficult to secure an airway. This can be a particular problem in small children. Endotracheal intubation is, thus, preferably performed on admission, before severe edema develops. On the other hand, unnecessary intubation and sedation can worsen a patient's condition. The decision to intubate should be made carefully by an experienced clinician.[6]

Definite indications for endotracheal intubation are:

1. Deep facial burns - especially of the mouth and oropharynx. Development of edema may impair patency in the near future.
2. Massive body burns, especially in the presence of circumferential chest burns, as ventilatory support is needed.
3. Severe inhalation injury.

Flow chart 10.1: Algorithm for management of smoke inhalation


Three types of smoke inhalation
• Carbon monoxide intoxication
• Inhalation injury above the glottis
• Inhalation injury below the glottis
Establish and maintain airway and adequate ventilation
Patient is conscious, no airway or ventilation problems no deep facial burn
Give 100% O2 by mask obtain blood gases, CO-Hb
One of the following is present: Unconsciousness Deep facial burn Hypoventilation Hypoxia on mask O2
Intubate (use tube size 7 or >) Diagnose and treat CO-Hb Begin 100% O2 to maintain O2 saturation >95% and displace CO-Hb. Clinically assess lower airways function. Obtain baseline chest X-ray. Aggressive suctioning for bronchorrhea. Use bronchodilators for bronchospasm. Consider chest wall escharotomy for 3° circumferential burn
CO-Hb is normal
Give O2 to maintain O2 saturation >90% Monitor for pulmonary problems
CO-Hb is raised
Give O2 until CO-Hb < 7% reserve hyperbaria for severest cases which are not responding to O2 alone
Begin standard Rx for smoke exposure
Continuous airway assessment: Aggressive pulmonary toilet Close monitoning of gas exchange Controlled fluid Rx Do not use prophylactic antibiotics or corticosteroids
Transfer to burn center

A patient with inhalation injury can be closely monitored for upper airway obstruction without a tube, preferably with the head elevated at 30°, only if intubation will be technically possible later. However, if anatomic distortion from face and neck burns is increasing to a point where safe intubation might soon be precluded, the procedure is carried out immediately. If edema distorts the anatomy, intubation using fiberoptic bronchoscope should be resorted to by an experienced clinician. **Prolonged and multiple attempts at intubation can further aggravate the hypoxia and may precipitate cardiac arrest.** So in the absence of expertise, the option of a tracheostomy should be exercised even if the incision has to be made through burnt skin (Fig. 10.9).

Once a definitive airway is secured, continuous administration of humidified oxygen is indicated to maintain adequate oxygenation as well as to assist clearing the airway of inspissated products of combustion. If the patient's hemodynamic condition tolerates, elevation of the head end of the bed by 30 degrees is helpful in reducing neck and chest wall edema. The early addition of bronchodilators, usually by aerosol, is especially advantageous in managing the bronchospasm seen after chemical injury. Nebulisation with Heparin + Saline helps in limiting the alveolar damage. After a secure airway is established, the need for ventilatory support is assessed. Blood gases are often misleading as far as saturation is concerned as the carboxy-hemoglobin is the confounding factor. The pH and bicarbonates can, however, guide therapy.

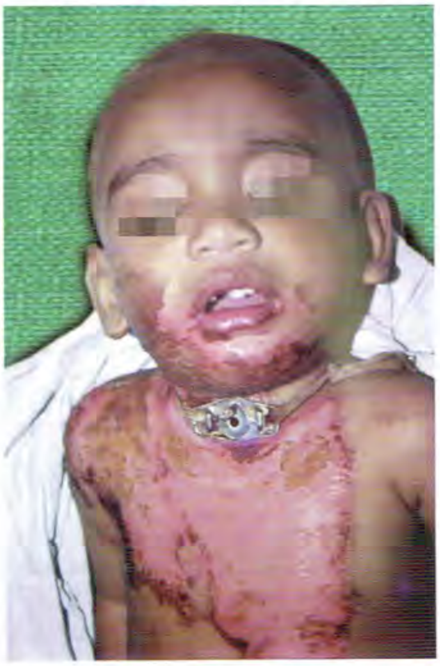


Fig. 10.9: Tracheostomy for laryngeal edema due to smoke inhalation

B – Breathing is evaluated for depth of respiration and effort. Detection of wheezing or rales suggests either inhalation injury with bronchospasm or aspiration of gastric contents. In a patient with burn, the respiratory effort and hence the gas exchange may be inadequate due to:

1. ***Mechanical restriction of breathing***—Deep dermal or full thickness circumferential burns of the chest can limit chest excursion and prevent adequate ventilation. This may require escharotomies.
2. ***Blast injury***—If there has been an explosion, blast lung can complicate ventilation. Penetrating injuries can cause tension pneumothorax and the blast itself can cause lung contusions and alveolar trauma and lead to adult respiratory distress syndrome.
3. ***Smoke inhalation***—The products of combustion, though cooled by the time they reach the lungs, act as direct irritants to the lungs, leading to bronchospasm, inflammation and bronchorrhea. Patients may need a period of ventilation, as this allows adequate oxygenation and permits regular lung toilet.
4. ***Carboxyhemoglobin***—Carbon monoxide binds to deoxyhemoglobin with 40 times the affinity of oxygen. It also binds to intracellular proteins, particularly the cytochrome oxidase pathway. These two effects lead to intracellular and extracellular hypoxia. **Pulse oximetry cannot differentiate between oxyhemoglobin and carboxyhemoglobin and may, therefore, give normal results.** However, blood gas analysis can reveal metabolic acidosis and raised carboxyhemoglobin levels but may not show hypoxia. Treatment is with 100% oxygen, which displaces carbon monoxide from bound proteins six times faster than does atmospheric oxygen. Patients with carboxyhemoglobin levels greater than 25-30% should be ventilated. It takes longer to shift the carbon monoxide from the cytochrome oxidase pathway than from hemoglobin. Hence, oxygen therapy should be continued until the metabolic acidosis has been corrected.

C – Circulatory status is evaluated with pulse rate and blood pressure and when indicated venous access is established with two wide bore intravenous lines in upper limbs in unburnt area. Initial fluid of choice is crystalloid. Colloids should be considered in patients not responding to crystalloids. **Profound hypovolemia is not the normal initial response to a burn.** If a patient is hypotensive then it may be due to delayed presentation, cardiogenic dysfunction or an occult source of blood loss (chest, abdomen or pelvis). In patients with circumferential deep partial or full thickness burns of extremities and electrical injury, assessment of distal circulation is mandatory for detection of **compartment syndrome.** The circulatory compromise can occur later as edema develops after initiation of fluid resuscitation. On securing vascular access, blood samples should be collected and sent for blood counts, packed cell volume, estimating urea and electrolytes, blood group and clotting profile.

Secondary Survey

The burn specific secondary survey should include (1) determination of the mechanism of injury (2) evaluation for the presence of inhalation injury and carbon monoxide intoxication (3) examination for corneal burns (4) consideration of the possibility of abuse and (5) detailed assessment of the burn wound. Besides evaluation of **A,B,C.** as detailed above, the secondary survey includes the following.

D—Neurological disability—All patients should be assessed for responsiveness with the Glasgow coma scale; they may be confused because of hypoxia or hypovolemia.

E—Exposure with environment control—Whole of the patient should be examined (including the back) to get an accurate estimate of the burn area and to check for any concomitant injuries. Burn patients, especially children, easily become hypothermic. This may lead to hypoperfusion and deepening of burn wounds. Patients should be covered and warmed as soon as possible.

F—Fluid resuscitation—The resuscitation regimen should be determined and begun. This is based on the estimation of the burn area. A urinary catheter is mandatory to monitor urine output in all adults with burns covering > 20% of TBSA. Children's urine output can be monitored with external catchment devices or by weighing nappies provided the injury is < 20% of total body area. In children the interosseous route can be used for fluid administration if intravenous access cannot be obtained, but should be replaced by intravenous lines as soon as possible.

History: The history of a burn injury can give valuable information about the nature and extent of the burn, the likelihood of inhalational injury, the time of burn, the depth of burn and probability of other injuries. The exact mechanism of injury and any prehospital treatment must be recorded. A brief medical history should be taken, outlining previous medical problems, medications, allergies and vaccinations. Patients' smoking habits should be determined as these may affect blood gas analysis. Development of edema after initiation of fluid resuscitation may hamper the patient's ability to vocalise. Hence, detailed history should be obtained from the patient at the initial assessment. After completing initial assessment and initiation of resuscitation, the complete extent and depth of burn wound should be evaluated.

Fluid Therapy (See Appendix 1.1)

Fluid losses from the injury must be replaced to maintain homeostasis. The main aim of resuscitation is to maintain tissue perfusion to the zone of stasis and so prevent the deepening of burn. This is not easy, as too little fluid can cause hypoperfusion whereas too much may lead to edema causing tissue hypoxia. Burns covering more than 15% of TBSA in adults and more than 10% in children warrant formal resuscitation as physiological compensatory mechanisms may not be adequate to combat the fluid loss. There is no ideal resuscitation regimen and many are in use. All the fluid formulas are only guidelines and their success depends on adjusting the amount of resuscitation fluid against monitored physiological parameters. The greatest amount of fluid loss in burn patients is in the first 24 hours after injury. For the first eight to 12 hours, there is a general shift of fluid from the intravascular to interstitial fluid compartments.[3] This means that any fluid given during this time will rapidly leave the intravascular compartment. Colloids have no advantage over crystalloids in maintaining circulatory volume in this situation. Fast fluid boluses probably have little benefit, as a rapid rise in intravascular hydrostatic pressure will just drive more fluid out of the circulation. However, much protein is lost through the burn wound, so there is a need to replace this oncotic loss. Some resuscitation regimens introduce colloid after the first eight hours, when the loss of fluid from the intravascular space is decreasing. The following facts need to be considered while planning fluid resuscitation.

1. Burn patients may have substantially higher fluid requirements than other trauma patients depending on the surface area involved.
2. Fluid needs to be titrated to maintain adequate output and special care needs to be taken when myoglobinuria is present (Fig. 10.10).

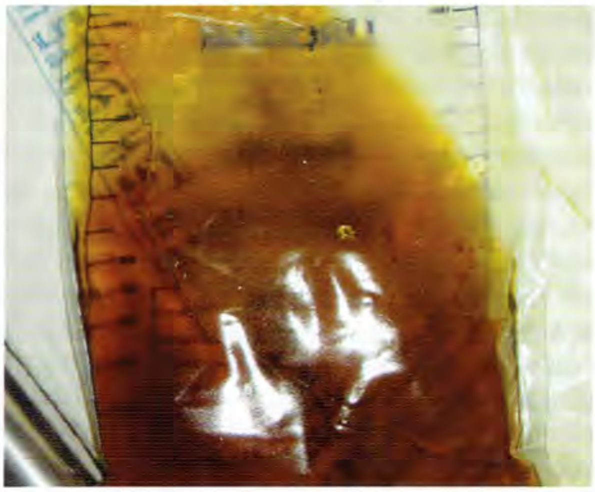


Fig. 10.10: Brownish urine suggestive of myoglobinuria

3. The greatest loss of fluids occurs in first 48 hours.
4. The fluid should be administered at constant drip rate and surges should be avoided. If indicated, aliquots of 250 ml of Lactated Ringer's may be administered.
5. The fluid volume for each patient should be adjusted according to urine output (30-35 ml per hour minimum) and hematocrit (32%). Insert urinary catheter.
6. In children, provision for the daily maintenance requirement (weight or surface area based formula) on top of the calculated amount should be made if Parkland formula is being used.
7. Sodium-salt solutions (crystalloids) are the essential components of fluid resuscitation.
8. The most appropriate fluid is Ringer lactate because its composition is the closest to that of extracellular fluid. Also lactate is a source of base due to its conversion to bicarbonate in the liver.
9. Fluids should be free of glucose (exception being small children), since glucose intolerance is characteristically present due to high circulating levels of stress hormones.
10. The time frame of fluid resuscitation is to be calculated from the time of burn and NOT from the time of initiation of therapy.

The resuscitation formulae most commonly used for fluid calculation are the Parkland formula, a pure crystalloid formula (4 ml × % burnt area × body weight in kg.), modified Parkland's formula or modified Brooke's formula (Table 10.5). The calculated volume is then given at different rates; first 8 hours - half of total to be given;

next 16 hours -half of total to be given;

next 24 hours - half of total given on first day.

Table 10.5: Resuscitation formulas (adult) for first 24 hours

Formula	*Crystalloids*	*Colloids*	*Daily need*
Parkland	4 ml/kg/% TBSA burn	None	None
Brooke (modified)	2 ml/kg/% TBSA burn	None	2.0 L
Galveston	5 L/m^2 burned area	None	1.5 L/m^2 BSA

Any fluid already given should be deducted from the calculated requirement.

Lactated Ringer's solution without dextrose is the fluid of choice except in children under 2 years, who should receive 5% dextrose Ringer's lactate.[7] Early work by Baxter and by Shire established the basis for modern protocols for fluid resuscitation.[8] They showed that edema fluid in burn wounds is isotonic and contains same amount of protein as plasma and the greatest loss of fluid is into the interstitial fluid compartment. It led to successful clinical trial of Parkland formula.

Colloid use is controversial. Some units introduce colloid after 8 hours, as the capillary leak begins to shut down, whereas others wait until 24 hours. Fresh frozen plasma is often used in children and albumin or synthetic high molecular weight starches are used in adults. Generally at the end of 24 hours, colloid infusion is begun at a rate of 0.5 ml× total burn surface area (%) × body weight (kg) besides maintenance crystalloid (usually dextrose-saline) infusion. The end point to aim for is a urine output of 0.5-1.0 ml/kg/hour in adults and 1.0-1.5 ml/kg/hour in children.

High tension electrical injuries require substantially more fluid (up to 9 ml × % burn area × body weight) in the first 24 hours and a higher urine output (1.5-2 ml/kg/ hour). Inhalational injuries also require more fluid.[3]

The above regimens are merely guidelines to the probable amount of fluid required. This should be continuously adjusted according to urine output and other physiological parameters (pulse, blood pressure and respiratory rate). Investigations at intervals of four to six hours are mandatory for monitoring a patient's resuscitation status. These include packed cell volume, plasma sodium, base excess and lactate.

The debate on the choice of resuscitation fluid has been on-going. Different fluids are expected to cause varying degrees of plasma volume expansion (Table 10.6). But, it was shown that changes in plasma volume were not related to the type of resuscitation fluid in first 24 hours, concluding that colloid solutions should not be used in first 24 hours until capillary permeability returns to near normal. Others have argued that normal capillary permeability is restored somewhat earlier (6-8 hours after burn) and colloids could

Table 10.6: Preload expanding properties of standard fluids

Fluid	*Osmolarity (mOms/L)*	*Colloid Osmotic Pressure*	*pH*	*Distribution Space*	*Ratio of fluid infused to intravascular expansion*
5% dextrose	250	0	4	Total body water	8:1
Normal saline	308	0	5	Extracellular	3:1
Ringer's lactate solution	270	0	6.5	Extracellular	3:1
Hypertonic lactated saline (250 mEq Na^+/L)	450	0	6-7	Extracellular expansion by fluid shifts from cells	<3:1*
6% albumin	250	20-24	6-7	Intravascular	1:1
Dextran 70	300-303	20-40+	6-7	Intravascular expansion by fluid shift	<1:1

* Depends on tonicity of fluid and plasma osmolarity

be started earlier.[9] Moncrief and Pruitt studied hemodynamic effects of fluid resuscitation in burns culminating in Brooke Formula which recommended use of 0.5 ml of colloid / % of burn / Kg body weight and has now been modified to all crystalloid resuscitation in first 24 hours. Efforts to avoid wrong calculation of resuscitation fluid have led to usage of Burns wheel[10], Palm-top computer [11] and Resuscitation Burn Card[12] etc. at some burn centers. There is continuing debate over the merits of colloid versus crystalloids for the resuscitation of major burns.[13] The crystalloid resuscitation is widely accepted. Hypertonic saline solution has theoretical advantage and has been shown to decrease net fluid intake[14] and decrease edema.[15] No significant difference was found in volume requirement or weight gain in patients over 20% TBSA burns, randomized on hypertonic saline or lactated Ringer's solution for resuscitation.[16] Some have found an increase in renal failure with hypertonic saline.[17] According to Berger et al, Hypertonic saline solutions, whether with dextran or not, have shown no advantage over the classic Ringer's lactate solution.[18] When using hypertonic saline, care must be taken to avoid serum sodium concentration greater than 160 mEq/dl.[19] Some burns unit use a modified hypertonic solution of sodium bicarbonate added to lactated Ringer's solution with success.[20] Some studies favor administration of protein-based colloids during the resuscitation period for major burns, starting in the early period after injury. These studies claim more beneficial effect than potential side-effects.[21] The results with albumin are varied. Some studies show that use of albumin does not reduce mortality when compared with crystalloids [22, 23], while other claims higher mortality rate with liberal use of albumin.[18] According to Cochran et al, there was no increase in mortality with albumin.[24] There was no reduction in multiple organ dysfunction score with albumin.[25]

According to Berger et al, artificial colloids, particularly gelatine, remain a useful tool in patients with major burns and hemodynamic instability and can be given as early as 6 hours after injury. [18] Chen et al studied utility of Hydroxyethyl starch in burns resuscitation and claimed that it could partially substitute plasma and be used in fluid resuscitation, but plasma protein replenishment should be emphasized.[26] The use of Composite Sodium Lactate and Sorbitol (CISS) in resuscitation fluid during shock stage of the patients with major burns can be beneficial to the replenishment of blood volume, control of shock, promotion of diuresis and subsidence of edema and can also provide electrolytes and energy, without the influence on the level of blood glucose.[27] Some found no added advantage with fresh frozen plasma and recommended its use only for specific coagulation disorders[24] while according to others, it is useful for resuscitation of burns. High dose (66 mg/kg/hour) intravenous ascorbic acid (vitamin C) in early phase of fluid resuscitation has been found to be advantageous.[28,29] This group showed reduced weight gain, wound edema, improved oxygenation and reduced requirement of resuscitation fluid volume.[29]

The ideal burn resuscitation formula does not exist. Whichever formula is used, patients must be monitored closely and the fluid resuscitation individualized according to the response. Patients with delay in resuscitation, associated trauma, inhalation injury or alcohol abuse may require greater than predicted quantity of fluid. The goal is to maintain urine output in the range of 0.5 to 1 ml/kg/hour for adults and 1 to 1.5 ml/kg/hour in children. In patients with fluid requirements greater than 150% of that predicted by formula, the addition of colloid at 12 hours can reduce total fluid requirements and burn edema. Early placement of pulmonary artery catheters can be useful in patients with known myocardial dysfunction, age greater than 65 years, severe inhalation injury or fluid requirements greater than 150% of that predicted by formula.[30]

Gastrointestinal fluid resuscitation[31] by oral and rectal route has been tried for burn patients in peripheral areas.

For burned children, the commonly used formulae are modified to account for changes in surface area to mass ratios. These changes are necessary because a child with a burn requires more resuscitation fluid per kilogram as compared to an adult (See Appendix 1.2).[32]

In Galveston formula the daily fluid requirement for a child with burn is augmented to 2 L/ m^2 of total body surface area against 1.5 L/m^2 for an adult.

Intravenous Access

A peripheral vein catheter through unburnt skin is the preferred route for fluid administration. A central line or pulmonary artery line is only occasionally required to monitor the patient during the initial resuscitation period and is removed as soon as possible. The possibilities for intravenous access are:

Choices for Venous Access

- First choice: Peripheral vein; unburnt area
- Second choice: Central vein; unburnt area
- Third choice: Peripheral vein; burnt area
- Worst choice: Central vein; burnt area

Monitoring lines are required primarily for the elderly patient or the patient with severe heart disease. A high complication rate has been reported with central catheters in burn patients as a result of infection.

Common Pitfalls in Initial Fluid Resuscitation

1. **Initial Under Resuscitation:** The largest fluid shifts occur in the first several hours post burn. Initial crystalloid infusion based on a formula will often be inadequate if shock state is present due to delay in initiation of resuscitation. More volume is needed. A volume expander such as a non-protein colloid or protein solution or hypertonic saline can be very useful along with isotonic crystalloid for successful resuscitation.
2. **Initial Over Resuscitation:** Aggressive fluid infusion is now frequently initiated in most emergency rooms with placement of large bore lines. The practice of "wide open" fluid infusion during early assessment and transport regardless of hemodynamic response is to be avoided. The excess fluid cannot be compensated for by decreasing infusion rate in subsequent hours becau the extra fluid is already out of the vascular space.
3. **Striving for Ideal Numbers:** It is quite possible achieve ideal numbers (pulse less than 120, blood pressu more than 100, urine output 0.5 to 1 ml/kg body weig hour) in a young patient with a moderate-siz uncomplicated burn. In a massive burn (more than 6(total body surface), especially in the presence inhalation injury or in the elderly, attempts at pushi fluids beyond reasonable amounts to achieve "id numbers" only accentuate edema-induced complicatio Adequate perfusion is not the same as "ideal number While planning the treatment, possibility of pulmon and chest wall edema-induced complications need to considered.
4. **Consideration of Fluid alone for Treatment Impaired Perfusion:** Hypovolemia from burn-induc fluid and protein shifts is the predominant cause hypoperfusion and fluid infusion is the treatme However, two other processes must be considered. T first is impaired cardiac output due to an increasing me airway pressure, particularly from a noncompliant ch wall burn. Ventilator adjustments, chest w escharotomy, elevation of the head of the bed to decre chest edema and the use of an agent, such as low-d dopamine, to assist fluids can be very helpful maintaining perfusion. Secondly, cardiac depressi from a deep body burn is well described, particularly the elderly. Supplementation of fluids, both crystall and colloid, with a beta agonist can help reverse downward course reflected by continually increasi fluid requirements and decreasing perfusion.
5. **Use of Urine Output alone as Monitor for Volu Restoration:** Although usually a reliable indicator renal perfusion, factors such as high plasma alcohol glucose levels often lead to an inappropriately high uri output relative to the status of tissue perfusion. T value of the urine output must be considered to unreliable under these circumstances.
6. **Failure to Secure Intravenous Lines:** As ede develops, the intravenous catheter may slip out of vein into the surrounding tissue. Not only does this le to transient hypovolemia, but finding a new site wh edema is present can be extremely difficult. A soluti is to secure the intravenous line with sutures.

Monitoring

In a burn patient no single monitor of perfusion can considered to be a completely reliable indicator of tiss oxygenation (perfusion). Therefore, several standard hemodynamic monitors and laboratory tests should be utilized.

Physiologic Measurements

Oxygenation: Use of pulse oximeter is standard for a major burn.

Baseline Body Weight: The baseline weight is used to estimate the initial fluid replacement and can be obtained by history or be estimated.

Arterial Pressure: The increased sympathetic tone characteristic of this early period makes arterial pressure an insensitive measure of volume status. However, a minimal level of perfusion pressure (more than 80 mm Hg) must be maintained. Thus, blood pressure monitoring is necessary.

An arterial line may be required if:

1. Patient is hemodynamically unstable.
2. Extremities are burned and sphygmomanometric pressure cannot be obtained manually.
3. If frequent blood gas analyses are required.

The arterial catheter should be placed through unburnt skin and should be removed as soon as possible.

Pulse Rate: Tachycardia is inevitable in early post burn period due to hypovolemia and catecholamine release from tissue trauma and pain. The degree of tachycardia can be very useful for determining adequacy of volume replacement. The exception would be an elderly or the patient with pre-existing heart disease in which the heart rate cannot increase in proportion to the stimulus. In most patients**: a pulse rate of less than 120/minute usually indicates adequate volume while more than 130/minute usually indicates more fluid is needed.**

Urine Output: The status of renal blood flow is usually an accurate reflection of the adequacy of systemic perfusion during this early phase of injury. A urine output of 0.5 to 1 ml/kg body weight/hour normally reflects adequate renal blood flow, assuming there are no factors such as alcohol, hyperglycemia or mannitol that alter the relationship between renal blood flow and urine output.

Intake-Output: What goes in and what comes out should be carefully tabulated. Intake may far exceed output during this phase as edema develops.

Blood Gases: The monitoring of arterial oxygen and carbon dioxide tension is required to judge the adequacy of ventilation. The measurement of pH and acid-base balance is extremely useful for the assessment of tissue oxygenation. A base deficit during this phase usually reflects impaired tissue oxygenation due to hypovolemia or carbon monoxide toxicity (also cyanide).

Electrocardiographic Monitoring: Arrhythmias are not common in the young patient as long as oxygenation is adequate. They become a major concern in the patient older than 45 years as a result of the response to burn stress. ECG monitoring is also indicated in patients with electrical injury.

Body Temperature: The burn patient is very prone to hypothermia during this early period, especially with infusion of cold fluids. A decrease in temperature can lead to further hemodynamic instability and impaired perfusion.

Central Venous Pressure (CVP): The central venous pressure in a large burn at this stage is usually low, 0 to 5 cm H_2O, even with adequate fluid resuscitation. Therefore, it can be very dangerous to use an arbitrary value of central venous pressure as an endpoint of resuscitation. Too much or too little fluid can be infused based on an arbitrary value.

Pulmonary Artery Wedge Pressure: The majority of young patients, even with massive burns, do not require the use of these measurements for initial resuscitation. A selected group of patients can benefit from this measurement. These include: 1. Elderly or patients with pre-existent heart disease with large burn or smoke inhalation. 2. Young patient with massive burn who is not maintaining perfusion despite fluid intake well in excess of predicted.

Cardiac Output, Mixed Venous Oxygen Tension: The primary objective of fluid management is to maintain adequate tissue oxygen delivery. The direct measurement of cardiac output (cardiac index) can assist in the determination of oxygen delivery. A cardiac index in excess of 2.5 L/min/m^2 is considered normal for an un-injured person. The value for mixed venous oxygen tension (PvO_2) however, can greatly assist in this determination.

Association of mixed venous gas

- $PvO_2 > 35$ mmHg: oxygen delivery adequate
- $PvO_2 > 30\text{-}35$ mmHg: oxygen delivery marginal
- $PvO_2 < 30$ mmHg: oxygen delivery inadequate

Laboratory Measurements (See Appendices 1.3 to 1.7)

Hemoglobin and Hematocrit: Baseline hematocrit and hemoglobin is useful for monitoring although changes in

Table 10.7: Hematocrit changes after large burn

Finding	*Cause*
Hematocrit increased	Plasma volume decreases while circulating red blood cell volume remains relatively constant
Hematocrit normal	Normalization of blood volume has occurred. Decrease in both plasma and red blood cell volume, the latter from, e.g. hemolysis
Hematocrit decreased	Hemolysis from prolonged heat exposure, with only plasma volume replacement. Major loss of blood from non-burn injury with only plasma volume replacement. Pre-existing anemia, hypervolemia (unlikely)

their values may not accurately reflect changes in blood volume due to the selective loss of the plasma component of blood (Table 10.7).

White Blood Cell: Initial white blood cell counts may be high, normal or low depending on the magnitude of the stress response and white cell sequestration into the burn. Low WBC count in association with inhalation injury is indicative of poor prognosis.

Electrolytes: Since the initial losses are primarily plasma, the Na^+, Cl^-, K^+ values remain relatively constant despite hypovolemia and vary mainly as a result of the type of resuscitation fluid used. K^+ increases in case of severe hemolysis or renal impairment. The HCO_3 content varies depending on the status of perfusion and acid-base balance.

Creatinine and Blood Urea Nitrogen: Baseline values are helpful to define normal status.

Plasma Proteins: A marked decrease in plasma proteins occurs early post burn.

Plasma Myoglobin: The plasma value of myoglobin is obtained in very deep burns, especially electrical burns. Myoglobin, released from deeply injured muscles, affects renal function. A higher urine output should be maintained (2-5 ml/kg/hour).

Prothrombin Time, Partial Thromboplastin Time and Platelets: An initial value during this period is useful for determining whether clotting factors may be needed. It is not common to have the need for replacing clotting factors and platelets during the first 36 hour period unless a prolonged shock state has initiated disseminated intravascular coagulation or pre-existing liver or hematologic disease is present.

Chest radiograph: An initial chest radiograph should obtained in all patients with inhalation injury.

Initial Management of Burn Wound

After ensuring airway patency, adequacy of respiration initiation of fluid resuscitation, attention is directed complete evaluation of the extent and depth of the b wound. Till such time, care should be taken to av hypothermia due to exposure. Based on the depth involvement, the burn wound is classified into **Superfici Superficial partial thickness, Deep partial thickn** and **Full thickness burn** (Figs 10.11 to 10.15). Burn de has an impact on healing time, the need for hospitalizat and surgical intervention and the potential for s development. Although accurate classification is not alw possible initially, the causes and physical characteristics burns are helpful in categorizing their depth (Table 10. Use of LASER, vital dye, ultrasound or thermography estimation of burn depth is expensive and cumbersom Visual estimation by experienced eyes is the most practi approach.

Wound care: For an acute burn which is to be transfer to a burn center, cling film is an ideal dressing as it prote the wound, reduces heat and evaporative losses, contr pain and does not alter the wound appearance.[6] This perm accurate evaluation by the burn team later. The details burn wound care are discussed elsewhere in this volu Only an outline is given here for sake of completion.

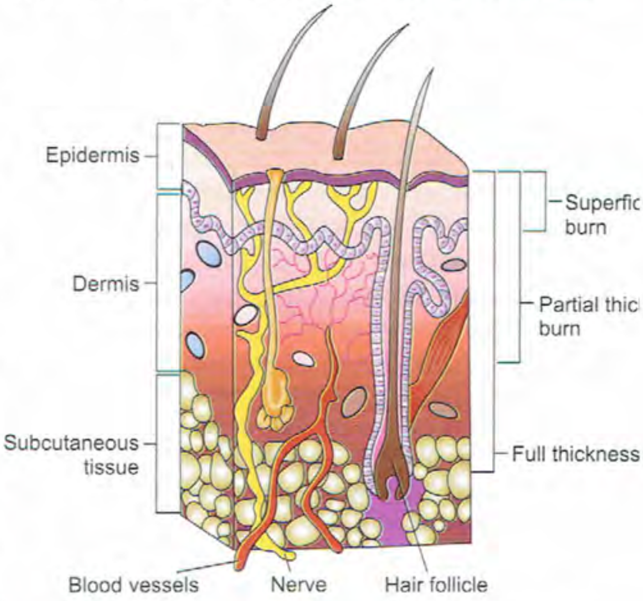


Fig. 10.11: Line diagram showing depth of burn injury

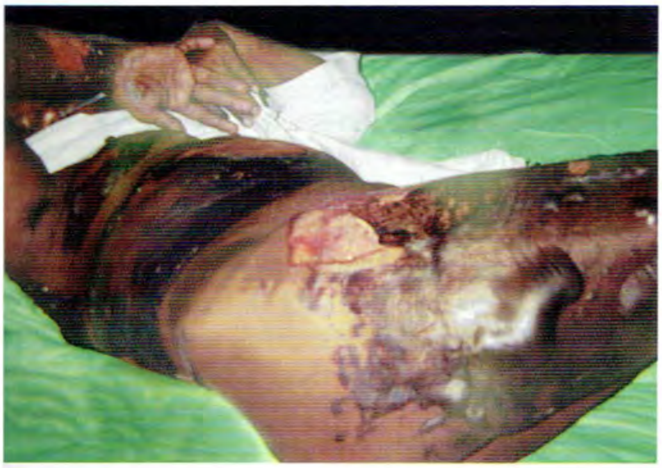


Fig. 10.12: Superficial partial thickness burn with blister

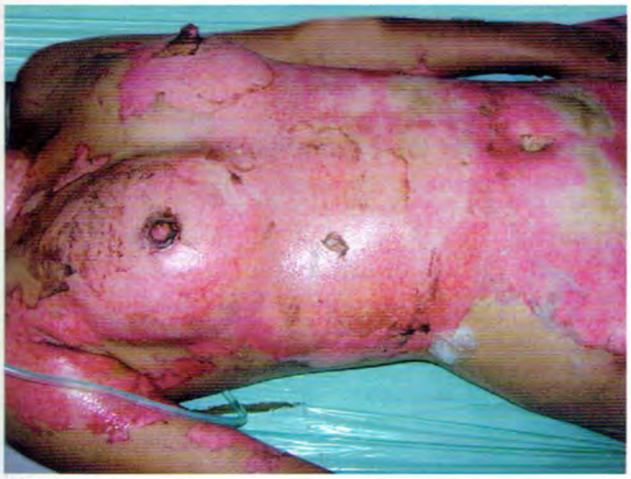


Fig. 10.13: Superficial partial thickness burn after debridement of dead skin

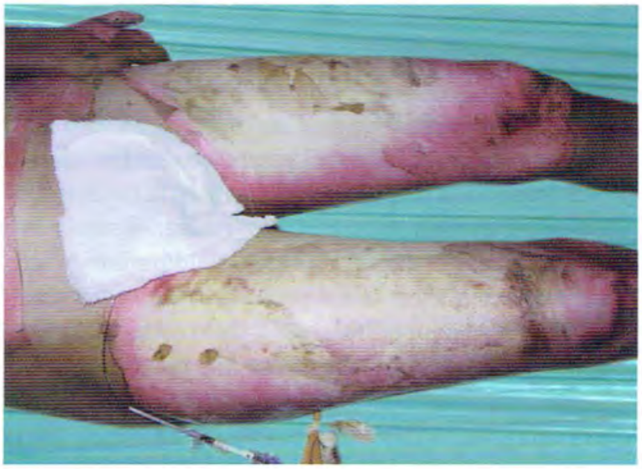


Fig. 10.14: Deep partial thickness burn

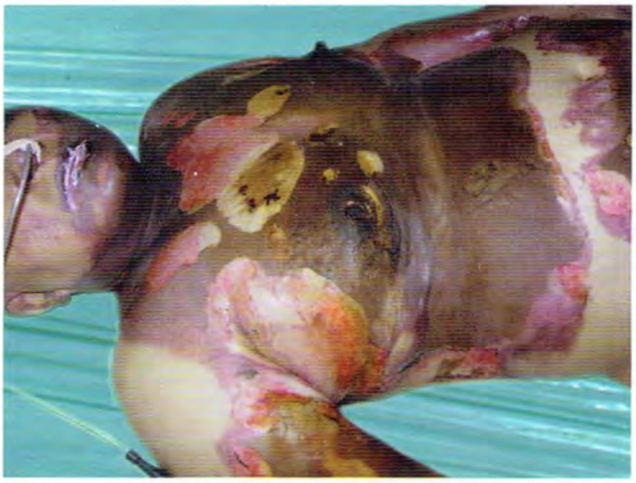


Fig. 10.15: Full thickness burn

Table 10.8: Classification of burns based on depth

	Characteristics				
Classification	*Cause*	*Appearance*	*Sensation*	*Healing time*	*Scarring*
Superficial burn	Ultraviolet light, very short flash (flame exposure)	Dry and red; blanches with pressure	Painful	3 to 6 days	None
Superficial partial-thickness burn	Scald (spill or splash), short flash	Blisters; moist, red and weeping; blanches with pressure	Painful to air and temperature	7 to 20 days	Unusual; potential pigmentary changes
Deep partial-thickness burn	Scald (spill), flame, oil, grease	Blisters (easily unroofed); wet or waxy dry; variable color (patchy to cheesy white to red); does not blanch with pressure	Perceptive of pressure only	More than 21 days	Severe (hypertrophic) risk of contracture
Full-thickness burn	Scald (immersion), flame, steam, oil, grease, chemical, high-voltage electricity	Waxy white to leathery gray to charred and black; dry and inelastic; does not blanch with pressure	Deep pressure only	Never (if the burn affects more than 2 percent of the total surface area of the body)	Very severe risk of contracture

For in-patients, the protocol followed at the author's unit is as follows:

A. Partial thickness burn wound: Wounds are cleaned with 0.5% chlorhexidine solution and normal saline. Blisters are deroofed and loose skin removed for ease of dressing, except for palmar blisters (painful), unless these are large enough to restrict movement. Wounds over face and hands and all burns in children are covered with collagen sheet if the patient is received within 6 hours of burn injury (Fig. 10.16). Once collagen is firmly adherent, secondary dressings are not needed. The author uses banana leaf dressing with 1% Silver-sulfadiazine (SSD) for dressing of all other partial thickness wounds.[33] This is covered with sterile Gamgee cotton pads and bandaged firmly (Fig. 10.17).

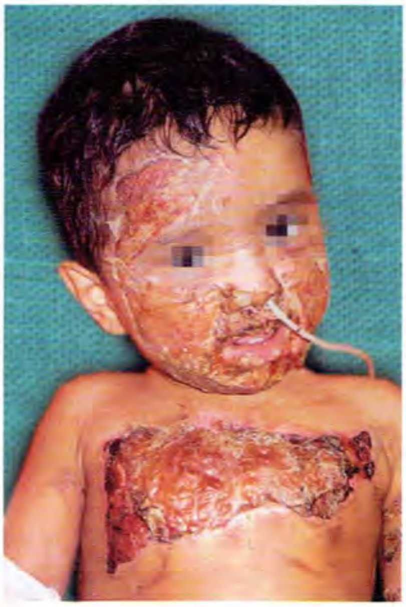


Fig. 10.16: Superficial partial thickness burn with collagen application

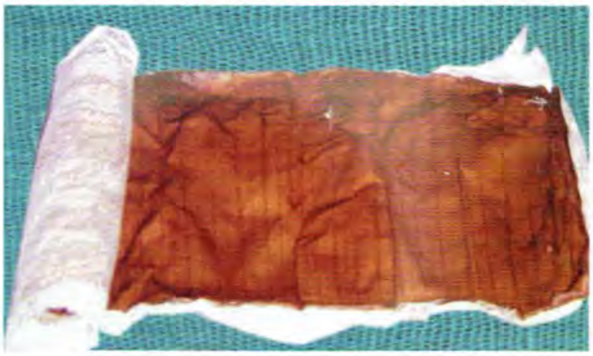


Fig. 10.17: Autoclaved banana leaf dressing

B. Full thickness burn wounds: In the author's unit, if early excision is planned, full thickness burn wounds receive exposure treatment with application of povidine-iodine ointment two or three times a day after cleaning of the wound with 0.5% Chlorhexidine solution. If early excision is not feasible, the wounds are dressed with banana leaf dressing with povidine-iodine ointment as the topical agent. At most other places the wounds are dressed with 1% SSD.

C. Infected wounds: Exposure treatment with povidine-iodine cream in the authors unit. Many other units prefer closed dressing with antimiciobial agent as per organism grown.

Early Surgical Management

Surgical procedures such as fasciotomy/escharotomy may be needed early in patients with full thickness circumferential burns of extremities or chest wall.

Impaired Chest Wall Compliance

Respiratory excursion can be markedly impaired by a circumferential deep partial or full thickness burn of the chest wall. The loss of elasticity in the burns of the chest wall markedly increases the work of breathing. As more subeschar edema develops, significant respiratory effort is required just to maintain adequate gas exchange. Any process that compromises the necessary increase in inspiratory force and muscle activity, such as hypoxia, hypovolemia, pain or sedation, accentuates the severity of lung dysfunction. Symptoms may not be clearly evident until edema development peaks at about 10 to 12 hours. The first clinical evidence of the chest wall restrictive defect is often labored breathing followed by a rapid respiratory deterioration, particularly in the patient who is not receiving ventilatory support. Clearance of secretions can be impaired due to the inability to generate a hyperinflation. In the combined chest burn and inhalation injury, it is very difficult to distinguish the degree of impairment in total lung compliance due to the increased airway edema and bronchospasm compared with that due to the impaired chest wall movement. An extremely deep burn results in tissue contraction due to desiccation, making the chest wall tight even before edema develops.

Patient with **non-circumferential** full thickness or partial thickness burn is monitored closely for evidence of respiratory restriction. Escharotomies are usually not required in a partial thickness burn unless the edema is so massive that the burned skin is tight. For **circumferential** full thickness burn, chest wall escharotomy is performed early. Even with an escharotomy, the restrictive process can be of such magnitude that hypoventilation is clearly evident. In these patients, endotracheal intubation and positive-pressure ventilation should be initiated before obvious pulmonary deterioration.

Impaired Distal Perfusion and Need for Escharotomy: A full thickness burn tissue has complete loss of elasticity. Hence, as subeschar edema develops under the burnt tissue, the tissue pressure increases. Fluid resuscitation leads to the development of burn wound edema and further swelling of the tissue beneath this inelastic burnt tissue. With circumferential full thickness burn of extremity, this increase in tissue pressure initially decreases the venous return. This further augments the tissue pressure and leads to impairment of arterial flow causing ischemia (Fig. 10.18). Initially, limbs at risk should be elevated and observed. Perfusion to the distal extremity should be closely monitored. Pain and color are unreliable indicators of perfusion in the presence of a burn.[3] A warm extremity invariably indicates good flow, but a cool skin does not always indicate that the problem is due to proximal constriction. Hypovolemia may well be the cause. Use of simple hand held Doppler equipment provides valuable information about perfusion. Evidence of ischemia mandates escharotomy (Flow chart 10.2). Both of these situations require escharotomy, division of the burn eschar. Only the burnt tissue is divided, not any underlying fascia, differentiating this procedure from a fasciotomy. Unless decreased blood flow is evident in the accident and emergency department, the escharotomy should be performed in the burn center after expedious transfer. In general, finger escharotomies should be performed in a burn center only because of the associated potential morbidity.

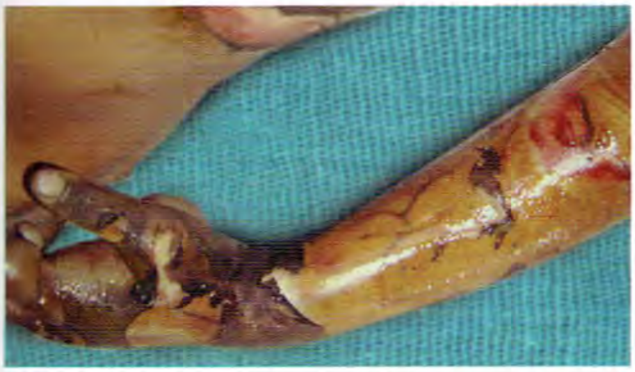


Fig. 10.18: Circumferential full thickness burn of forearm leading to ischemic damage to hand

Principles of Escharotomy

1. The escharotomy incisions should be located as shown in (Fig. 10.19). Incisions are made along the midlateral or medial aspects of the limbs, avoiding any underlying structures. For the chest, longitudinal incisions are made at each mid-axillary line to the sub costal region. The lines are joined up by a chevron incision running parallel to the sub costal margin.[3] This creates a mobile breast plate that moves with ventilation.
2. Anesthesia is not required as the full thickness burn is insensate.
3. The procedure should preferably be performed in operation theater, but can be performed bedside with all aseptic precautions.
4. The incision should be deep enough to allow separation of the edges permitting bulging of viable tissue.
5. Pain and significant bleeding is indicative of excessively deep incision.
6. Escharotomy must extend completely through length of burnt tissue crossing the joints till healthy skin is reached.
7. Escharotomies are best done with electrocautery, as they tend to bleed. Use of microcrystalline collagen to pack the incision site can help control punctate bleeding. Larger vessels usually require suture ligatures or cautery.
8. Dressing should be applied using a topical agent and nonadherent wound cover.
9. Monitoring of distal perfusion should be continued after escharotomy and if the impairment persists, existing escharotomies should be extended or additional escharotomies should be performed.

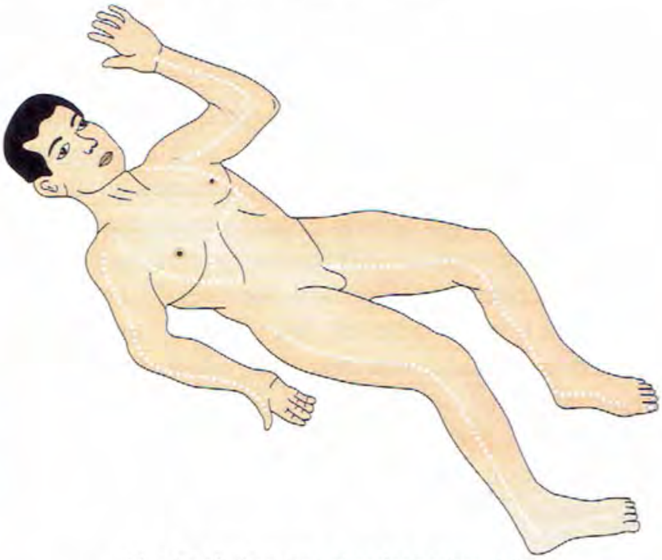
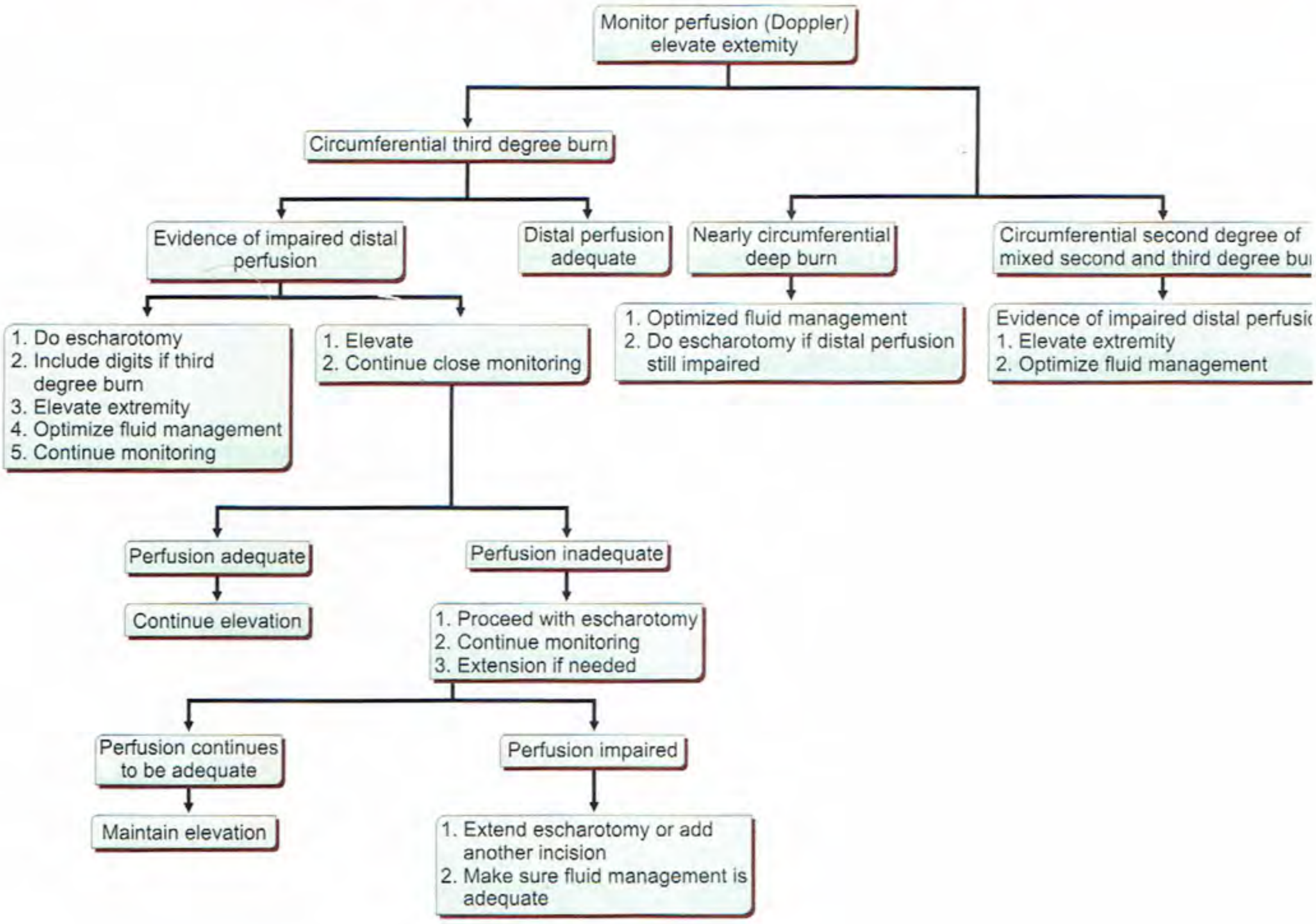


Fig. 10.19: Common sites for escharotomy

Table 10.10: Algorithm for management of circumferential extremity burn


Monitor perfusion (Doppler) elevate extemity
Circumferential third degree burn
Evidence of impaired distal perfusion
Distal perfusion adequate
Nearly circumferential deep burn
Circumferential second degree of mixed second and third degree bu
1. Do escharotomy
2. Include digits if third degree burn
3. Elevate extremity
4. Optimize fluid management
5. Continue monitoring
1. Elevate
2. Continue close monitoring
1. Optimized fluid management
2. Do escharotomy if distal perfusion still impaired
Evidence of impaired distal perfusi
1. Elevate extremity
2. Optimize fluid management
Perfusion adequate
Continue elevation
Perfusion inadequate
1. Proceed with escharotomy
2. Continue monitoring
3. Extension if needed
Perfusion continues to be adequate
Maintain elevation
Perfusion impaired
1. Extend escharotomy or add another incision
2. Make sure fluid management is adequate

Fasciotomy

Treatment of the high-voltage electrical burn victim should include prompt initiation of aggressive fluid resuscitation and serial assessment of distal vascular integrity. Development of compartment syndromes should be anticipated. Deeper muscle groups sustain the greatest injury. Urgent surgical intervention for fasciotomies and muscle compartment explorations under regional or general anesthesia is often indicated. Intraoperative exploration, decompression and debridement are an essential part of early treatment. Serial technetium-99m stannous pyrophosphate muscle scans has been shown to be useful in assisting determination of progressive muscle damage.[34] If fasciotomies are delayed, development of compartment syndrome and acute renal failure is imminent along wi development of gangrene of the affected extremity (Tab 10.9).

Besides early fasciotomy, fluid resuscitation deman attention. If the urine is rose-pigmented fro hemochromogens, the urine output should be maintained 100-125 ml/hr (1-2 ml/kg/hour) in adults or twice the norm hourly rate in infants and young children, until gross pigme is cleared (Figs 10.20A and B). The urine can be alkalinizi by IV administration of sodium bicarbonate to preve myoglobin precipitation. In severe injuries, IV mannitol (12 gm in an adult) is given in addition to aggressive flu replacement to promote an osmotic diuresis.[34] Early u of other diuretics is contraindicated.

Table 10.9: Key points for escharotomy and fasciotomy

- Remove constricting objects
- Immediate elevation of burned extremities
- Perform early escharotomies on circumferential full thickness burn
- Consider early fasciotomies for electrical burns involving extremities.
- Decreasing Doppler flow signal with adequate blood pressure or tissue pressure more than 25 mmHg means escharotomy/fasciotomy is needed.
- Escharotomy is performed without anaesthesia while fasciotomy requires regional anesthesia.
- Escharotomy must extend completely through the burnt tissue.
- Hemostasis is obtained with pressure, cautery or microcrystalline collagen.

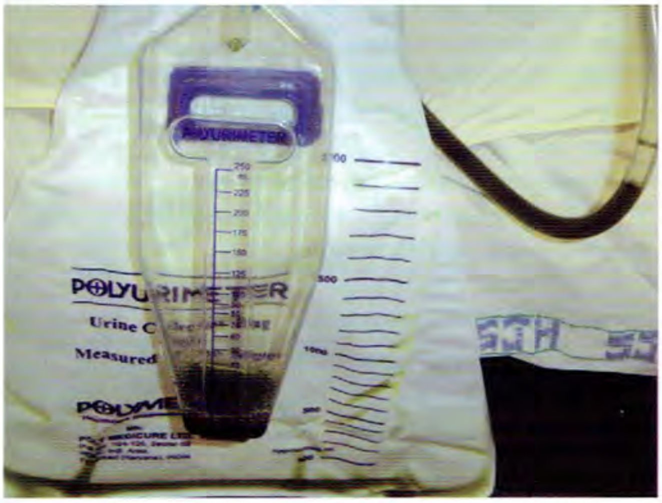


Fig. 10.20A: Dark reddish black urine due to myoglobinuria suggestive of renal insult, following electric burns with gangrenous changes in hand

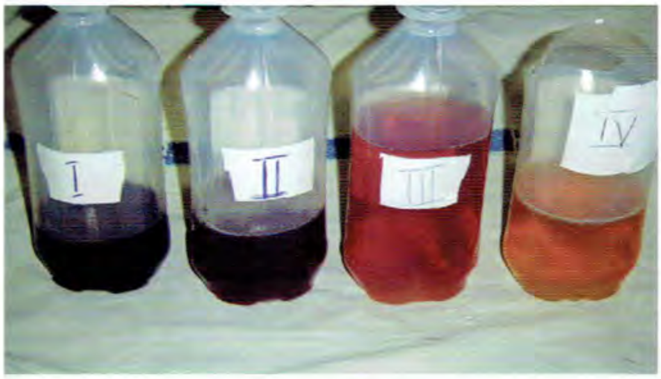


Fig. 10.20B: Rapid fluid infusion with I/V mannitol and sodium bicarbonate till urine gradually clears to its normal color–This prevents hemochromogens from blocking the renal tubules

INITIAL MANAGEMENT OF BURNS INVOLVING SPECIAL AREAS

The face, ears, hands, genitals and the feet have functional and cosmetic significance that far exceeds their size. Burns of these areas need special attention and this should be initiated early during the treatment.

Face Burns

- Suspect inhalation injury in patient with burns of face.
- After proper cleansing, apply collagen sheet in a patient with superficial partial thickness burns, if seen within 6 hours of injury.
- Deep partial or full thickness wounds may be treated by exposure with silver sulfadiazine cream smear or may be covered with face mask of nonadherent dressing and Gamgee cotton pads.
- 30° propped up position. Insert Ryle's tube for feeding.

Eye Burns

- History of mode of burns and eye examination.
- Observe for singed eyelashes, eyebrows, eyelid edema, conjunctival lacerations, corneal opacity, any foreign body, pupil size and reaction to light.
- If chemical injury is suspected, irrigate the eye with Normal Saline 0.9% for 15-20 minutes.
- Application of ophthalmic antibiotic ointment 6-hourly reduces risks of corneal drying and infection.
- Cover the eye with eye shield and NOT with gauze and cotton pad.
- Repeat examination daily to look for corneal ulcer, abscess or other complications
- In the event of corneal injury or with severe burns of the eyelids, an ophthalmologist's consultation should be obtained.
- Early tarsorrhaphy should be avoided as it increases lid deformity and prevents serial examinations of the corneal surface.

Ear Burns

- History of burn injury and examination of ear.
- Examine the auricle for edema, tenderness and look for discharge from canal.
- If blisters are present, puncture them and expel the fluid out but avoid removing any skin which is still firmly attached

- Avoidance of any pressure on the burned auricle is essential (no pillows). Pressure is the biggest co-factor in the production of chondritis[35] (Fig. 10.21).
- Apply paraffin gauze into the canal and apply povidone iodine ointment on the burnt pinna. Wrap the ear with nonadherent dressing and Gamgee cotton pad and apply mastoid bandage gently.
- Most ear burns respond well to conservative treatment, although occasionally immediate coverage of exposed cartilage with a temporoparietal facial flap and skin graft may be required to salvage the pinna.

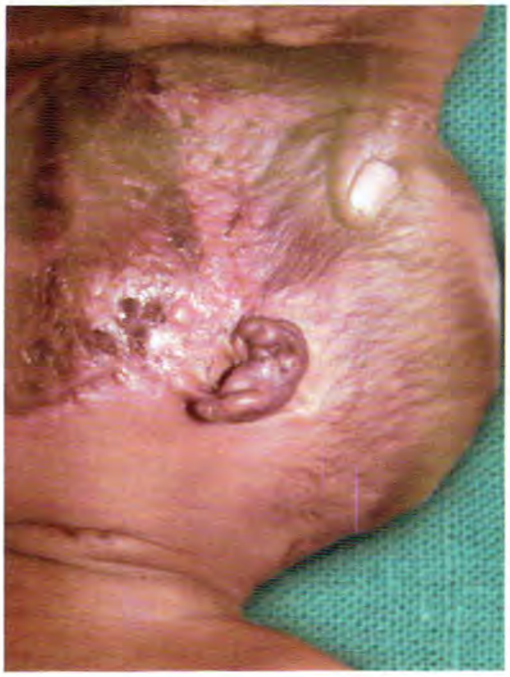


Fig. 10.21: Chondritis following ear burn

Hand Burns

- Hand burns assume a high priority from the onset of care.
- During the first 24-48 hours, adequate blood flow must be ensured. The consistency, the temperature and the presence of pulsatile flow detectable by Doppler ultrasonography monitoring of the digital pulp should be regularly performed. If any doubt exists, escharotomy or fasciotomy should be performed.
- While dressing a hand burn, each finger should be wrapped individually with nonadherent tulle grass and web spaces should be separated.
- The hands should be splinted in a position of function: the metacarpophalangeal joints at 70-90°, the interphalangeal joints in extension, the first web spa open and the wrist at 20° of extension.[35]
- The hands should be elevated to minimize edema and therapist should perform range of motion exercises twi daily.
- Deep dermal and full-thickness burns should underg early excision and sheet autograft closure.

Feet Burns

- They require specific care; so admit the patient wi burns to feet (especially circumferential burns).
- Assess the depth of the burns (Fig. 10.22). Strict lim elevation of the involved extremity.
- Look for capillary refill to assess vascularity.
- Do escharotomy early to avoid gangrene of the dist part in patients with circumferential burns. Perfor primary excision with skin grafting whenever possibl
- Apply antimicrobial cream and paraffin gauze in we spaces.
- Throughout the day encourage the patient for active well as passive physiotherapy of ankle joint to preve early contracture formation.

Perineal Burns

- Admission of this patient is necessary for specific car
- Assess extent and depth of the burns. Look for the ski loss and edema.
- Per-urethral catheterization is essential to avoi contamination by urine.
- Apply a layer of Silverex ointment over burnt area an cover it with double layer of paraffin gauze and Gamg or keep it exposed.

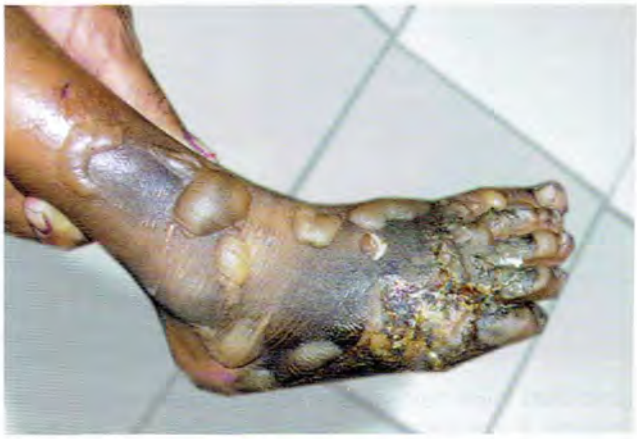


Fig. 10.22: Burns of foot

- Administer appropriate antibiotic cover including anaerobic organisms, according to protocol.

PAIN MANAGEMENT

Burn injuries cause one of the most intense and prolonged pain. Burn pain is difficult to control because of its unique characteristics, its multiple components and its changing patterns over time [36] (Table 10.10). Cleansing wounds, changing dressings and providing physical therapy involve repeated manipulations of painful sites. The sometimes excruciating pain, along with extreme intra- and inter-individual variability in severity, complicates the task of providing analgesia to burn patients.

Table 10.10: Unique characteristics of burn pain

- Three major components:
  1. ***Background:*** At rest in burned areas and at skin graft donor sites
  2. ***Breakthrough:*** During simple activities such as changing position, turning in bed, or walking
  3. ***Procedural:*** During wound care procedures that must be performed daily or even several times a day; can reach excruciating levels
- Is highly variable among patients and within each individual
- Can last for weeks or months
- Does not decline over time except toward the end of hospitalization

Current research suggests that early vigorous analgesic intervention is crucial to prevent the adverse consequences of uncontrolled pain. For example, the burn patient who associates dressing changes with agonizing pain is difficult to treat, even with massive doses of opioids. Prompt, aggressive analgesia is critical to avert the cycle of pain, anxiety and more pain and to minimize adverse psychological effects of pain commonly encountered in burn patients.

Acetaminophen (Paracetamol) or NSAIDs with opioids can be a basic treatment for burn pain.[36] Traditional analgesic regimens usually neglect or ignore the inflammatory component of burn pain, but it is extremely important, especially during early treatment. Traditional NSAIDs are not recommended in burn patients who are scheduled to undergo extensive excision and grafting procedures because their antiplatelet effects may increase blood loss. However, this class of drugs may help patients who require minimal skin grafts to reduce pain from superficial burn areas or at donor sites (normal skin areas where skin is harvested for grafting). Newer NSAIDs, the cyclooxygenase-2 (COX-2) inhibitors, offer analgesia with few side effects, including minimal inhibition of platelet function.[36] Although the effects of COX-2 inhibitors on wound healing remain to be demonstrated, such medications may constitute a major advance in the treatment of burn pain. These drugs can be introduced early during treatment to reduce peripheral and central sensitization, decrease opioid requirements and reduce side effects.

Closed dressing over the partial thickness burn wounds contribute significantly to pain relief as the exposed nerve endings are protected from external stimuli. In patients with major burns (i.e. with > 25 % TBSA burns) analgesics should be administered by intravenous route during early management (i.e. 48-72 hours after injury) as absorption by intramuscular route is unpredictable and oral administration may not be possible. For minor burns, the pain medication may be administered by oral route.

The medications and their recommended dosages are as follows:

1. **Morphine:** It should be administered only after resuscitation has been initiated and following precautions should be observed
   a. Do not give if patient is asleep. Do not give for sleep, give only for pain.
   b. Do not give if patient is in shock or in sepsis.
   c. Monitoring of ECG, pulse, respiration and oxygen saturation is essential.
   d. Do not give if respiratory rate is less than 20 or oxygen saturation is less than 95 %.

   In adults, patient controlled analgesia (PCA) probably provides maximum pain relief. Morphine infusion is given intravenously using PCA pump with total dose 15 microgram/kg/hour as continuous infusion and can be combined with patient administered boluses.

   Morphine can be administered 0.03 mg/kg every 4 hours intravenously after stabilizing the patient with resuscitation.

   Oral morphine (0.1 mg/kg every 4 hours) can be given after 48 to 72 hours post burn. This can be used to provide relief from both background as well as procedural pain experienced during bath, dressing or physiotherapy.

2. **Buprenorphine:** An alternative for Morphine, Buprenorphine should be administered by observing same precautions as morphine. The dose is 300-600 micrograms slowly intravenously over 10-15 minutes every 6 hours for adults. It is not recommended for children under 6 months of age. For children, the

recommended dose is 3-6 micrograms/kg of bodyweight, 6 hourly.

3. **Butorphenol:** 1-4 mg every 3-4 hours, slowly intravenously.
4. **Pentazocine:** Pentazocine Lactate 30 mg intravenously 3-4 hourly is recommended for adults. It is not recommended for children below 1 year of age. Above 1 year of age, the dose recommended is 0.5 mg/kg every 6 hours.

Besides the above mentioned opioids, the following NSAIDs can be used to provide effective pain relief.

1. **Diclofenac:** 1mg/kg intravenously 6 hourly as slow infusion.
2. **Tramadol:** 50-100 mg intravenously slowly or as infusion every 4-6 hours upto a maximum dose of 600 mg/day.
3. **Ibuprofen:** Intravenous ibuprofen is under investigation. The oral preparation can be used in children as well as adults in minor or moderate burns on admission and in major burns after the resuscitation is complete. The recommended dose is 10 mg/kg 4 hourly.
4. **Paracetamol:** 15 mg/kg can be used orally for procedural pain relief.

Orally administered Acetoaminophen (15 mg/ kg) and Codeine can be combined along with opioids for effective pain relief in major burns.

Agents such as Ketamine, Propofol, Fentanyl, Alfentanyl, etc. should be used for controlling procedural pain under supervision of an anesthetist and after complete resuscitation in a patient with major burn.

Anxiolytics such as Lorazepam or Midazolam may be combined with opioids or NSAIDs especially for relief of procedural pain. The recommended dose is 0.03 mg/kg slow intravenously.

Adequate pain relief is an important but often neglected aspect of early management of burn. If attended to appropriately in the initial period, it contributes significantly to sustained compliance of the patient with various treatment procedures.

EARLY REHABILITATIVE CARE

Once the patient's survival has been assured, function and cosmesis become the biggest factors for subsequent quality of life. However, if efforts to achieve these goals are not begun early in the initial management, the ultimate outcome may be less than desirable despite successful resuscitation and burn wound closure. Rehabilitation of the burn patient should begin during the acute resuscitation period an continue until the patient's scars mature and occupatio resumes. Rehabilitation plans are carried out principally b the Occupational and Physical Therapists.

There are principles which must be applied soon aft burn to ensure the earliest and optimal rehabilitation of th patient. Rehabilitative care should commence on the da of the injury and the goals of burn patients' rehabilitatio are:

1. To limit or prevent loss of motion.
2. To prevent or minimize anatomic deformities.
3. To prevent loss of lean muscle mass.
4. To ensure return of the patient to work or normal activit as soon and as completely as possible.

Much consideration must be given to a program o rehabilitation for the burn patient and every patient needs a individually tailored plan of care. There are four principle for the rehabilitation of the burn patient:[37]

- The program should start early, preferably on the da of injury.
- A program of care should avoid prolonged periods o immobility and any body part that can move freely shoul be moved frequently.
- Range of motion exercises should be started on the da of injury.
- There should be a planned program of daily activity an rehabilitative care. The plan should be reviewed dail as rehabilitative needs change.

On admission, plans should consider the prevention o skin and muscle contracture and anatomic deformity. The institution of such plans should be individually tailored. Early standing and ambulation and participation in daily livin activities are important and all extremities should be activel moved frequently throughout the day. Proper positionin is essential for the prevention of contractures. It has no been uncommon in the past for burned patients to develop contractures of both burned and non-burned joints. The incidence of contractures in healing patients has been significantly reduced through the use of frequent active o appropriate passive motion exercises and proper positioning. Burned patients and their families should be taught the importance of early active exercise and proper positionin during rest and sleep. Standing and ambulation should be instituted as soon as possible. Such exercise can reduce the loss of muscle mass and help stimulate the appetite. Frequent standing and ambulation also reduces the risks o pressure necrosis. Additionally, requiring the patient to ge out of bed and sit in a chair for some part of the day increases respiratory tidal volumes and the patient's sense of normalcy. This provides a great psychological boost to the patient.

Detailed account of rehabilitation activities is outside the purview of this chapter and is discussed elsewhere in this volume.

MEDICAL RECORDS

Most of the burn patients are medicolegal cases and are to be informed to the law enforcement authorities and meticulous medical records are of significant value in the court of law. Besides the routine record of history and clinical examination, the following information should be sought and entered in the medical records of the patient.

1. Record the time of burn, exact incident and persons present at the site.
2. Name and relation of person bringing the patient to hospital and providing information.
3. Record details of pre-hospital treatment, co-morbid conditions.
4. Record the actual incident in notes and not just–'scalds with hot tea' or 'flame burns with kerosene'.
5. If the patient wishes to change the statement, record the new statement and place signature with name, date and time. Inform the police constable on duty about the patient's wish to change statement.
6. Inform the expected prognosis and patient's condition to the close relatives and record it on the case paper with the signature of the relative and name with date and time.
7. The consultant should enter his/her observations and treatment decisions in the record in his/her own writing with signature, date and time.
8. Detailed orders including special instructions, wound management plan and monitoring instructions should be recorded.
9. Details of procedures performed during resuscitation and reports of investigations should be recorded.

SUMMARY

1. The early management of seriously burnt patients is similar to the management of other trauma victims. Life threatening injuries must be treated first, followed by secondary survey. Airway, breathing and circulation should be attended to on priority. High index of suspicion is required for inhalation injuries. Hypothermia should be avoided.
2. If the burn surface area is greater than 15 % in an adult and greater than 10% in a child, intravenous fluids should be administered to restore the circulating blood volume in order to maintain tissue perfusion and oxygenation.
3. Sodium containing crystalloid is the fluid of choice and the most useful indicator of fluid resuscitation is hourly monitoring of urine output.
4. Meticulous estimation of the extent and depth of burn is necessary for planning the wound management. Burns of special areas deserve attention. The rehabilitative measures should be initiated early.
5. Completeness of medical records is highly desirable. Systematic initial evaluation, methodical application of basic surgical principles of resuscitation and vigilant monitoring using simple clinical parameters leads to successful initial management of a burn patient and also lays the foundation for ultimate satisfactory outcome.

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Chapter

11

Respiratory Tract Injury in Burns

JC Suri, MK Sen

INTRODUCTION

Injury to respiratory tract due to burns was little appreciated prior to 1940s. A pivotal point in the understanding of pulmonary injury in burn patients happened to be the famous "Boston night-club fire" in 1942. Between 1945 and 1975, several innovations took place, which went a long way to recognize the respiratory injuries and treat these patients in a better and scientific way. In the last two decades, management has changed greatly and survival has become more likely.

Injury to the respiratory tract in a patient with burns can occur in several ways. Firstly, there may be respiratory tract involvement in pure thermal injuries. Second, there can be non-thermal involvement of the respiratory tract due to inhalation of either asphyxiant gases (like carbon monoxide and cyanide) or irritant gases (like chlorine, ammonia, oxides of sulfur and nitrogen). Anoxia is also another principal mode of injury in respiratory tract involvement due to inhalation. Inhalation injuries are reported in 19-28% of all burn patients. On the other hand, the incidence may rise to 58 % in victims who die.

PATHOGENESIS

The principal effects of inhalation injury include asphyxia and direct respiratory tract injury. Asphyxia can result from hypoxia or intoxication by carbon monoxide or cyanide. These factors can affect the patient either alone or in combination. Direct injury to the respiratory tract can involve the airway or the alveolar-capillary membrane. Involvement of the airway results in upper airway obstruction, tracheobrochitis, bronchiolitis or atelectasis. On the other hand, alveolar-capillary membrane injury can result in pulmonary edema due to high permeability of the membrane. Inhalation injury has now become one of the most important cause of death in burn patients. The mortality from smoke inhalation alone is low (0-11%). However, in combination with cutaneous burns, it may be fatal in 30 to 90% of patients. It has been recently reported that the presence of inhalation injury increases burn mortality by 20% and that inhalation injury predisposes to pneumonia. Pneumonia has been shown to independently increase burn mortality by 40% and the combination of inhalation injury and pneumonia leads to a 60% increase in deaths.

Cutaneous burns can result in pulmonary dysfunction, chest wall restriction (e.g. circumferential neck and chest burns) or upper airway compression. Quite often, a combination of smoke inhalation injury and cutaneous burns co-exist in the same patient.

Other co-morbid respiratory complications in a patient with burns include concomitant chest trauma and exacerbations in a pre-existing cardiopulmonary disease like cardiogenic pulmonary edema or chronic obstructive pulmonary disease. There may be a variety of nosocomial / iatrogenic problems in such patients. They include hydrostatic pulmonary edema (due to over-enthusiastic fluid resuscitation), pneumonia (aerogenous or hematogenous), sepsis-related permeability edema (acute respiratory distress syndrome), drug induced conditions (hypoventilation due to morphine or hyperventilation due to mafenide), pulmonary embolism and complications due to artificial airways and aspiration.

Direct injury to respiratory epithelium and alveolar macrophages by chemicals like aldehyde that are present in smoke can trigger inflammation. This occurs due to release of chemotactic factors as well as activation of phospholipase. The various chemotactic factors lead to priming of neutrophils; entrapment of these primed neutrophils in the pulmonary vasculature, in turn, leads to their activation and degranulation, resulting in release of proteolytic enzymes and oxygen radicals which cause lung edema. Activation of phospholipase, on the other hand, results in formation of prostaglandins (thromboxane A_2) leading to bronchoconstriction, hypoxic pulmonary vasoconstriction and lung damage. Airway obstruction and ventilation-perfusion mismatch also occur.

The pathological alterations induced by inhalation injury on lung host-defenses include mucosal burns, laryngeal obstruction, tracheobronchitis, paralysis of mucociliary apparatus, bronchorrhea, mucosal sloughing, bronchospasm, airway edema, atelectasis, phagocyte dysfunction, alveolitis and a decrease in pulmonary surfactant levels. The potential immunosuppressive mediators following thermal burns usually include bacterial endotoxin, prostaglandins (PGE2), endogenous glucocorticoids, cytokines (Tumor Necrosis Factor-alpha (TNF-α), interleukins, interferon), histamine, immune complexes, neuropeptides, autoantibodies and suppressor peptides.

Thus, a plethora of pathological effects of burn injury occurs on the host defenses. They can result from depression in cell-mediated immunity, activation (and subsequent depletion) of the complement system, suppression of phagocyte functions (macrophage and neutrophil), release of immunosuppressive mediators, increased consumption of immunoglobulins, depletion of fibronectin and serum opsonic activity and an increase in T-suppressor cell activity.

Pathophysiology of Lung Parenchymal Damage by Smoke Inhalation

Incomplete products of combustion present, for instance, in burning cotton, aldehydes and oxides of sulfur and nitrogen, hydrochloric acid and carbon monoxide, etc. are all responsible for damage to lung parenchyma. Upper airway damage, on the other hand, is mainly due to direct thermal injury and chemical irritation.

A gradually progressive edema of the tracheobronchial mucosa takes place over the initial 24 hours. In the tracheobronchial mucosa, areas of de-epithelialization are demonstrable; in the lower respiratory tract focal areas of congestion and edema, alternating with areas of collap
and pneumonia are seen. Lesions in the airway range fro
superficial desquamation of epithelium to focal necrosis a
formation of pseudomembranes comprising mucus, cellul
debris, fibrinous exudates, polymorphonuclear leucocyt
and clumps of bacteria. Tracheobronchial casts c
ultimately lead to respiratory obstruction. A significa
decrease in pulmonary compliance takes place due to increa
in extravascular lung water and pulmonary lymph flow.

Inactivation of surfactant results from inhalation inju
leading to extensive microatelectasis. Microvascular inju
with increased fluid flux to interstitium and alveolar spa
resulting in profound hypoxemia and acute respirato
distress syndrome (ARDS) can also ensue.

CLINICAL FEATURES AND DIAGNOSIS

The general impact of respiratory complications of smo
inhalation (airway injury, parenchymal injury and late on
problems) is significantly higher in cases who ha
concomitant cutaneous burns (Table 11.1).

Fiberoptic bronchoscopy helps to confirm the diagno
of inhalational injury. It can identify areas of airway oede
mucosal sloughing or charring or soot in the upper airwa
It is the current gold standard for diagnosis of inhalati
injury in major burn centers. Rigid laryngoscopy and flexi
nasopharyngoscopy are also helpful. In addition, repeat
bronchoscopies with lavage can also be done to help
cleaning mucosal plugs. Chest skiagrams are of mini
help in the initial period.

Xenon lung scans can evaluate alveolar air trappi
Simultaneous thermal and dye dilution methods can be us
to assess extravascular lung water and determine parenchy
fluid levels. Measurements of serum carboxyhemoglo
levels are of immense importance.

Table 11.1: Presenting symptoms and signs of inhalation injury

	Relative frequency (%)
Dyspnea	3-17
Hoarseness	13-61
Throat pain	10-13
Chest pain	32-41
Cough	9-76
Hemoptysis	6-18
Carbonaceous sputum	9-80
Cyanosis	6-15
Tachypnea	25-80
Stridor	5-33
Wheezing	13-47
Rhonchi	23-52
Crackles	23-35

A battery of diagnostic tests is judiciously utilized in the management of patients with involvement of respiratory system. They include objective measurement of asphyxiants in the blood (carboxyhemoglobin, cyanide), arterial blood gases, chest roentgenogram, endoscopic examination (laryngoscopy, nasopharyngoscopy and bronchoscopy), radio-nuclide scanning, and pulmonary function tests (spirometry).

Respiratory Complications of Smoke Inhalations

Common clinical presentations vary from patient to patient and depend to a large extent on the degree of burns. At one end of the spectrum are patients with smoke exposure without overt respiratory dysfunction and no respiratory injury with normal objective tests. There may be sub-clinical injury with either normal/abnormal objective tests. Various grades of clinically overt respiratory dysfunction can be present. At the other end, there may be cardio-pulmonary arrest or asphyxiant death at the scene of exposure. The various symptoms and signs include dyspnea, hoarseness, throat pain, chest pain, cough, hemoptysis, production of carbonaceous sputum, cyanosis, tachypnea, stridor, wheezing, rhonchi and crackles.

In less severe cases, there is an evidence of airway obstruction and decreased pulmonary compliance on pulmonary function testing. After several days of injury, endobronchial debris can lead to small airway obstruction and atelectasis.

Pulmonary edema is a sinister end result that can result from multiple causes. Acute respiratory distress syndrome (ARDS) is a common case scenario in a large majority of burn-patients undergoing treatment in tertiary care hospitals (Table 11.2). Common risk factors associated with the occurrence of ARDS include sepsis syndrome with or without shock, severe trauma (thoracic or extrathoracic), pneumonia (bacterial, viral), aspiration of gastric contents, disseminated intravascular coagulation (DIC) and near drowning. Occasionally, it may be associated with fluid overload, smoke or toxic gas inhalation, extensive surface burns, fat embolization, anaphylactic reaction to drugs and blood, neurogenic injury, toxic drugs and poisons. It is characterized by tachypnea, refractory hypoxemia, diffuse pulmonary infiltrates and reduced lung compliance. Over the last decade, several studies have suggested that survival rates for patients with acute lung injury (ALI) or ARDS may have improved.

The diagnosis of ARDS becomes highly probable in the presence of the following features: tachypnea, $PaO_2/FiO_2 < 200$, presence of known common risk factors, diffuse pulmonary infiltrates on chest X-ray and reduced lung compliance. Hematological disturbances (like coagulopathy, DIC and thrombocytopenia), multiorgan failure, ratio of bronchoalveolar lavage fluid protein to serum protein being more than 0.8 and a characteristic pathology are also typical of ARDS.

Smoke inhalation injury has been clinically graded on the basis of bronchoscopic criteria as follows:

Grade Features

0 No signs or symptoms referable to the respiratory tract. Absence of carbonaceous deposits, erythema, bronchorrhea or obstruction (No injury)

1 Signs and symptoms limited to the upper respiratory passages, including mild laryngitis. Minor or patchy areas of erythema, carbonaceous deposits in peripheral or distal bronchi (Mild injury)

Table 11.2: Diagnosis of ARDS

High Probability	*Moderate Probability*
Tachypnea	Tachypnea
$PaO_2/FiO_2 < 200$	$PaO_2/FiO_2 < 300\text{-}350$
Known common risk factors	Uncommon risk factors
Diffuse pulmonary infiltrates	Patchy or multiple segmental infiltrates
Reduced compliance	Near – normal compliance
Pulmonary hypertension	Normal pulmonary resistance
Coagulopathy(Thrombocytopenia, DIC)	Normal coagulation or only thrombocytopenia
Multiorgan failure	Only pulmonary involvement
High bronchoalveolar lavage (BAL)/serum total Protein ratios >0.8	BAL/serum total protein < 0.6
Large protein molecules found in BAL > 70A	BAL molecular protein radii < 70A
Characteristic pathology	No pathology specimen available

2. Evidence of involvement of the trachea and bronchi or abnormal signs in the lungs other than those of frank consolidation. Moderate degree of carbonaceous deposits, erythema, bronchorrhea, with or without compromise of bronchi (Moderate injury)
3. Dyspnea, cyanosis, wheezing or transient stridor or frank signs of consolidation. Severe inflammation with friability, copious carbonaceous deposits, bronchorrhea, bronchial obstruction (Severe injury)
4. Evidence of extensive pulmonary involvement or of obstruction to the airways necessitating tracheostomy or resulting in severe asphyxia. Evidence of mucosal sloughing, necrosis, endoluminal obstruction (Massive injury)

Major risk factors include house fires, explosions and other disasters. Direct thermal damage due to flame, liquid or steam inhalation is rare. Thus, the spectrum and time course of respiratory problems require special attention in all patients. Classical clinical predictors should be watchfully monitored and they include exposure characteristics (like closed-space setting, entrapment, unconsciousness, known inhaled toxin), respiratory signs and symptoms, presence of facial and cervical burns and production of carbonaceous sputum (Figs 11.1 and 11.2).

Asphyxia manifests as altered sensorium, coma, nausea, vomiting; upper airway obstruction as stridor, hoarseness dyspnea, sore throat, painful swallowing, respiratory area burns, flash exposure, pharyngeal edema/erythema; lower airway obstruction as dyspnea, tachypnea, wheezing, carbonaceous sputum and parenchymal injury as dyspnea, tachypnea, crackles. Resuscitative measures for cardiopulmonary arrest: successful cardiopulmonary resuscitation (CPR) (grade V); supportive measures for clinically obvio established respiratory dysfunction (grade II to IV) a accurate triage for exposure history [possible delayed-ons dysfunction (grade I)] should be promptly taken.

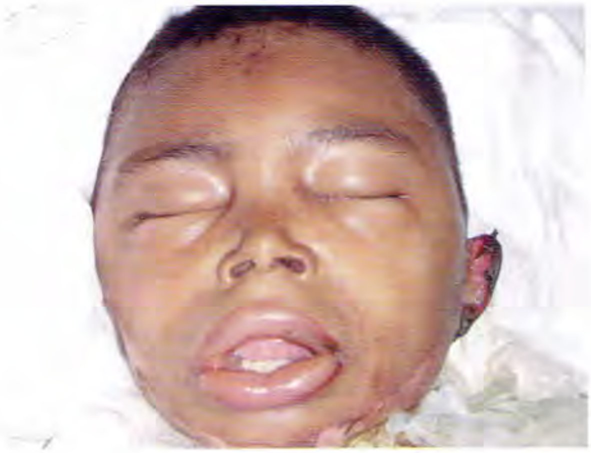

Fig. 11.1: Case with minimal burns of face but with signs of inhalation injury

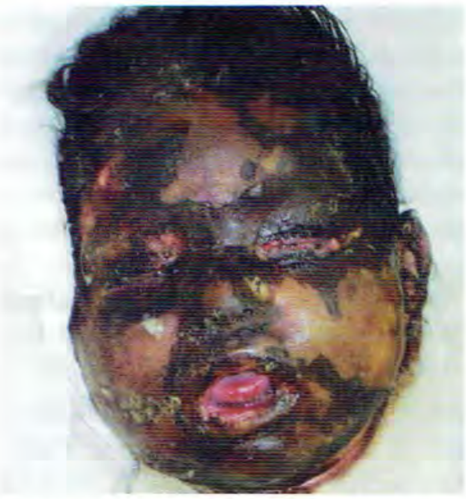

Fig. 11.2: A 3-year-old child with deep burns of entire face wit evidence of inhalational injury

MANAGEMENT

Management of Inhalation Injury in Patients without Cutaneous Burns

The management of inhalation injury in patients witho burns begins with a good history and examination of t accident scene. Circumstances of inhalation should examined (history of suicidal attempt); closed spaces (e. building, automobiles) result in more frequent and seve respiratory abnormalities. The severity of inhalation inju is also directly related to the duration of exposure. The lun can be an efficient means for the absorption of inhal toxicants, resulting in airway and pulmonary injury systemic toxicity. The type of smoke inhaled is also extremely important factor. Exposure to smoke fro polyurethane foam results in severe parenchymal inju Time elapsed between initial resuscitation and arrival hospital and presence of co-morbid disease(s) (chron obstructive pulmonary disease, COPD; coronary arte disease, CAD etc.) are also important determinants severity of diseases. A history of closed space fi production of carbonaceous sputum, facial burn, sing nasal hair, perioral burn, hoarseness, stridor or pharynge edema are important pointers to upper airway inju (Figs 11.1 and 11.2).

Airway maintenance is of prime importance. Early intubation is indicated in case upper airway burn is present. Airway edema increases over 12-18 hours. Nebulization with bronchodilators, heparin (10,000 units in 3 ml N-Saline every 4 hours), N-acetylcysteine (3-5 ml of 20% solution every 4 hours) are also very useful steps. Indications for endotracheal intubation include inability to protect airway, respiratory distress, hoarseness/stridor, dyspnea, 3rd degree facial burns, carbon monoxide (CO) poisoning and edema of supraglottic structures.

The management of respiratory burns has two primary components, namely, bronchial hygiene therapy and mechanical ventilation.

The thrust areas of bronchial-hygiene therapy are as follows:

1. Oxygenation of the patient with humidified oxygen so as to maintain $SpO_2 > 90\%$ (Fig 11.3).
2. Therapeutic coughing: either voluntary or induced by placing index and middle fingers over sternal notch.
3. Deep breathing exercises every two hours.
4. Therapeutic posturing and chest physiotherapy every 4 hours.
5. Turning the patient side to side every two hours.
6. Aerosolization of 3 ml of 20% N-acetylcysteine every 4 hours with a bronchodilator.
7. Aerosolizing 5000 units of heparin with 3 ml of normal saline every four hours.
8. Nasotracheal suctioning as and when required.
9. Encouragement of early ambulation by post burn day 5.
10. Culture of endotracheal secretions thrice a week.

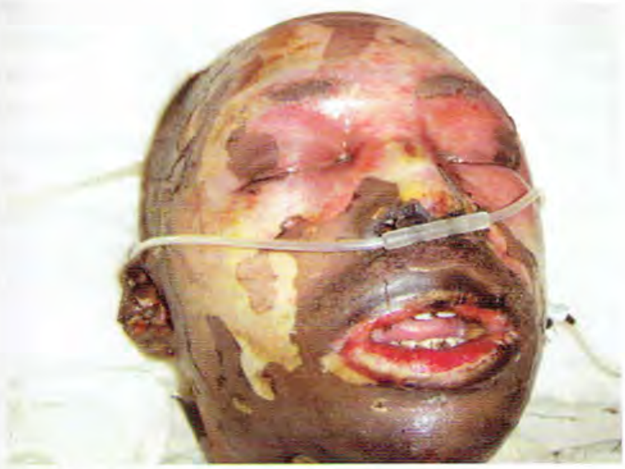

Fig. 11.3: Patient with facial burns with element of upper airway injury requiring only oxygenation by nasal prongs

Mechanical Ventilation

Mechanical ventilation is mainly initiated in the scenario of acute refractory hypoxemia as seen in cases of acute respiratory distress syndrome (ARDS) (Fig 11.4). Several modes of ventilation are available to the clinician. The mode used should be the one which is able to support oxygenation and ventilation. Additionally, the clinician should also be experienced in its use.

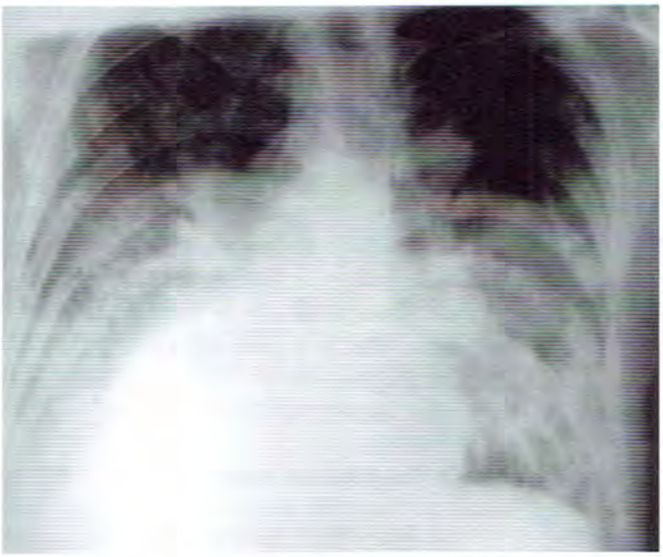

Fig. 11.4: X-ray chest showing ARDS

The ARDS Network study conducted by the National Heart, Lung and Blood Institute of US (NHLBI) recommends the use of low tidal volumes of 6 ml/kg of predicted bodyweight and a plateau pressure of 30 cmH_2O or less while using the volume-assist mode. If the patient becomes obstructed due to fibrin casts and develops an acute hypercapnia and hypoxia, pulmonary toilet should be done and change over to higher tidal volumes is considered. Initial flow rates of 40-100 liters per minute, inspiratory /expiratory (I: E) ratio of 1:1 to 1:3 and FiO_2 (fraction of inspiratory O_2) of 1.0 may be sufficient. Positive end expiratory pressure (PEEP) is applied to recruit lung units, elevate mean airway pressure and improve oxygenation. The level of PEEP varies with the disease process. Pressure-volume curves help in determining the optimal level of PEEP. The optimal level of PEEP is the one that results in the lowering of pulmonary shunt, improvement in arterial oxygenation, only a small change in cardiac output, A-V oxygen content differences or mixed venous oxygen tension.

The various modes of ventilation available are as follows:

a. Control mode, where the ventilator cycles automatically at a rate selected by the operator.
b. Assist-control mode, where the ventilator only assists every spontaneous breathing effort of the patient; a back-up control rate is set. Also set are tidal volume, inspiratory flow rate, flow waveform, sensitivity and control rate.
c. Synchronized intermittent mandatory ventilation (SIMV), where a pre-set number of ventilator–delivered mandatory breaths with pre-set tidal volume are delivered along with the facility of intermittent spontaneous breaths. It is usually a weaning mode.
d. Pressure control mode, where a ventilator breath of constant pressure (pressure limited, time-cycled and pressure triggered) is delivered. The inspiratory time, pressure limit and back up rate are pre-set by the operator. Tidal volume is dependent on compliance of chest and airway resistance. This mode can be used in ARDS.
e. Pressure support mode, is a pressure targeted, flow-cycled and patient triggered breath. The patient must have an intact respiratory drive. It is a useful weaning mode.

Newer Modes of Mechanical Ventilation

a. High frequency percussive ventilation (HFPV): It provides ventilation at low mean airway pressure and supports the lung-protective strategy of ventilation to reduce barotraumas. Sub-tidal volumes are delivered in a progressive stepwise fashion until a preset oscillatory equilibrium is reached and exhalation is passive. A high-frequency time-cycled pressure ventilator (VDR ventilator) is used. The pulsatile flow rate is set at 20 cm H_2O peek inspiratory pressure (PIP); the pulse-frequency (high rate) set at 500-600. The low respiratory rate is set at 15-20 and demand PEEP on 3 cm H_2O. The I: E ratio is set at 2:1. Settings of the ventilator can be adjusted later, based on clinical and arterial blood gas (ABG) parameters.
b. Inverse ratio ventilation: It involves the setting up of I: E ratio (inspiration to expiration ratio) greater than 1:1. It maintains a high mean airway pressure and high peak alveolar pressure; it prolongs inspiratory time to allow for recruitment of lung units in dependent areas of chest. Time-constant sedation and paralysis are always required for this mode.
c. Airway pressure release ventilation (APRV): It is a pressure-regulated mode of ventilatory support which allows for time-cycled decreases in pressure to facilitate elimination of CO_2.

Weaning and Discontinuation of Mechanical Ventilation

Attempts should be made to remove the patient from mechanical ventilation as early as possible. The criteria that should be followed before any such attempt is made are as follows:

1. Reversal of underlying cause of respiratory failure, namely improvement in respiratory burn/sepsis.
2. Adequate oxygenation as evidenced by $PaO_2/FiO_2 > 200$-250, PEEP requirement $\leq$ 5-8 cm H_2O, $FiO_2 \leq 0.4$-0.5 and pH > 7.25.
3. Hemodynamic stability or low-dose vasopressor requirement.
4. Presence of spontaneous inspiratory effort.

A spontaneous breathing trial with T-tube breathing for 30 minutes or pressure-support/continuous positive airway pressure (CPAP) of 1 hour should be administered. Respiratory mechanics should be assessed.

1. Respiratory rate < 24 in adults
2. Spontaneous tidal volume > 4 ml/kg
3. Expired minute volume < 15 liters/minute
4. Negative inspiratory force > 30 cm H_2O
5. Audible leak around endotracheal balloon cuff.

Racemic epinephrine aerosolization can help in alleviating any inspiratory stridor that may be present following extubation (due to mucosal edema).

Late Complications of Inhalational Injury

1. Tracheal stenosis due to tracheitis, tracheal ulceration and tracheal granuloma formation. It occurs in the region of the endotracheal cuff (sub-glottic region). It is best prevented by paying attention to tracheal tube-cuff pressure, particularly in the background of hypotension.
2. Obstructive/restrictive lung disease is occasionally observed following inhalation injury.

Spirometry, cardiopulmonary exercise testing and rehabilitation are important aspects in the after-care of these patients.

Carbon Monoxide Poisoning

Carbon monoxide (CO) is the most common gas liberated during fires. It is colorless, tasteless and has an affinity for hemoglobin that is 200 times that of oxygen. It causes reversible displacement of oxygen on the hemoglobin molecule. It interacts with the myoglobin of cardiac muscle and with mitochondrial cytochrome 3 oxidase complex leading to additional effects on cellular oxygen metabolism and organ function. When combined with hemoglobin (carboxy hemoglobin-COHb), it results in impaired oxygen delivery to the tissues and a decrease in mixed venous oxygen content. Clinical features may result from mild headache and confusion in lower degrees of exposure to coma, seizures and death in severe cases (Table 11.3). In a case of blood carboxy hemoglobin level of 5-10%, the symptoms include mild headache and confusion. Throbbing headache, blurred vision and flushing of the skin occur when it rises to 11-20% while at a level of 21-30%, the clinical manifestations include disorientation, nausea and impaired manual dexterity. Irritability, dizziness, vomiting and syncope occur when the levels rise to 31-40%. In case the levels go to beyond 50%, coma, seizures, respiratory failure and death may ensue.

Table 11.3: Signs and symptoms of CO intoxication

Carboxy hemoglobin level (in percent)	*Clinical manifestations*
5-10	Mild headache, confusion
11-20	Throbbing headache, blurred vision, flushing of the skin
21-30	Disorientation, nausea, impaired manual dexterity
31-40	Irritability, dizziness, vomiting, syncope
41-50	Tachypnea, tachycardia
50 and above	Coma, seizures, respiratory failure, death

The elimination of CO is dependent on the level of oxygen in blood (FiO_2) and the law of mass action. It occurs in two phases: (a) a rapid distribution to other tissues and (b) a slower excretion phase. The elimination half-life of CO while breathing room air is 250 minutes; at 100% oxygen, it is 40 minutes while at 2 atmospheres hyperbaric oxygen it is 30 minutes. At 2.5 atmospheres hyperbaric oxygen, the dissolved oxygen content of plasma alone (>5 ml/100 ml) is sufficient to meet the basal body oxygen requirements. The various neuro-psychiatric sequelae of CO poisoning include Parkinsonism, cortical blindness, temporospatial disorientation, deafness, memory changes, mental retardation and frank psychosis.

Patients with mild to moderate exposure (COHb < 30 %, no coma, no neurologic findings) are probably treated adequately with 100% oxygen. A patient who is comatose on admission, has a COHb level of greater than 30% or has an existing neurologic deficit, is at risk for permanent damage and a hyperbaric oxygen (HBO) chamber, if readily available, may benefit from a dive. The patient with associated problems (burns greater than 40% of body surface area (BSA), other injuries or airway compromise) should be considered for hyperbaric treatment only if there is no risk to life endangered by the delay in treatment of the other problems. Hyperbaric oxygen therapy can, at times, be associated with several complications. They include barotrauma to ears, aspiration, seizure, hypocalcemia, oliguria, metabolic acidosis, respiratory acidosis, dysarrhythmias and rarely cardiac arrest. Indications for HBO include altered mental status, loss of consciousness/cardiac arrest at scene, focal neurological deficits, seizures, syncope, ischemic chest pain, ECG changes, new dysarrhythmias, COHb>25%, COHb>15% with cardiovascular/cerebrovascular disease, pregnancy with COHb>10% or with signs of fetal distress.

Cyanide (CN) Poisoning

Cyanide poisoning is usually seen with burning of polyurethane in the construction and furniture industries. It acts by inhibiting the final step of oxidative phosphorylation by binding the cytochrome AA3 complex resulting in lactic acidosis and cellular asphyxia. Exposure to cyanide results in altered level of consciousness, dizziness, headache, tachycardia and tachypnea. Blood levels of CN should be measured and lethal levels range between 1.0 to 3.0 mg/L. Any patient with positive clinical criteria or blood CN levels greater than 0.20 mg/L should be treated. Cyanide is normally detoxified primarily by hepatic rhodonase and requires sulfate as a substrate. Treatment includes the following measures (3-step CN antidote package). Amyl nitrite is given by inhalation for 15-30 seconds each minute. Sodium nitrite (300 mg in 10 ml) is injected over 2-4 minutes with careful monitoring of blood pressure. These two steps produce methemoglobin (MetHb), which combines with cytochrome-bound-CN to yield cyano-MetHb. Sodium nitrite injection is given to keep MetHb levels below 40%. Finally, sodium thiosulfate (12.5 gm in 50ml of 50% dextrose) is injected over a 10-minute period. Vitamin B_{12} (hydroxycobalamine) 400 µg/Kg intravenously is given to produce inactive cyanocobalamine. Cyanide shunted to these minor pathways must eventually be detoxified by rhodonase.

Airway obstruction associated with inhalation injury must be treated with inhaled β_2-agonists (salbutamol, terbutaline), racemic epinephrine, aminophylline (0.5-0.9 mg/kg/hour as infusion), and anticholinergic drugs (ipratropium bromide). The incidence of proximal airway edema on the modern battlefield is likely to increase with the advent of enhanced blast and thermobaric weapons systems. Current military medical doctrine suggests that soldiers who are at risk of airway closure from edema should have a surgical airway provided. Tracheal burns caused by thermal or chemical injuries are uncommon. They can be difficult to manage. Acutely, they are often life threatening unless diagnosed quickly and managed properly. Tracheal burn injuries can lead to long-term stenosis, which requires specialized surgical techniques and chronic therapy, often with Montgomery T-tubes, for their proper management.

Impaired oxygenation: Other problems like (supplemental oxygen, mechanical ventilation with PEEP, ventilatory strategies like high frequency oscillatory ventilation (HFOV), airway control, hypoventilation, overhydration (limitation of fluid intake), airway secretions (bronchoscopic removal of secretions) should also be addressed simultaneously.

Management of Inhalation Injury in Patients with Cutaneous Burns

Cutaneous burns affect pulmonary function by release of circulatory factors and intravascular hypoproteinemia. The degree of pulmonary dysfunction depends on effective care of the burn site (wound-care). Basic principles of management are similar to those outlined in the section on care of inhalation injury without burns. Additional treatment of burn area aimed at stopping burn process, dressings and intravenous fluids are important measures. The presence of facial and pharyngeal burns should raise the suspicion of upper airway injury. Early endotracheal intubation in such cases is desirable (Figs 11.5A and B). Tacheostomy can be performed to avoid permanent damage to upper airway or vocal cords (Figs 11.6A and B). Patient should be weaned off the ventilator and tracheostomy tube as early as is possible (Fig. 11.7). Depolarizing muscle relaxants should be avoided. Stabilization of the endotracheal tube in patients with facial burns requires specialized skill. Controlled fluid management with close hemodynamic monitoring, mechanical ventilation with PEEP and chest wall escharotomies for full thickness thorax burns are other measures that may be helpful (Fig. 11.8). Management of delayed sequelae includes the treatment of pneumonia and burn sepsis. A combination of inhalation and cutaneous burn injury leads to increased incidence of pneumonia (46%) due to local pulmonary changes and global immunosuppression. Enteral glutamine administration has been found to be helpful in burn patients.

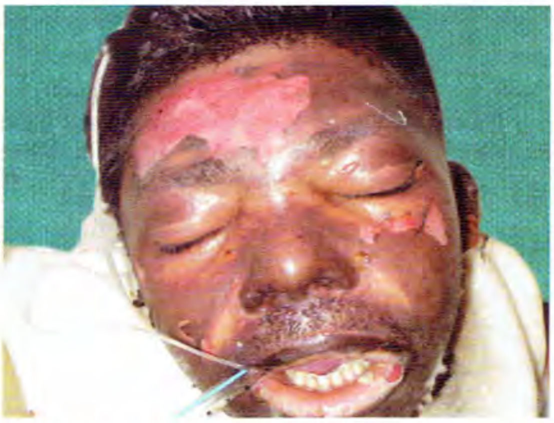

Fig. 11.5A: Patient with facial burns with endotracheal tube *in*

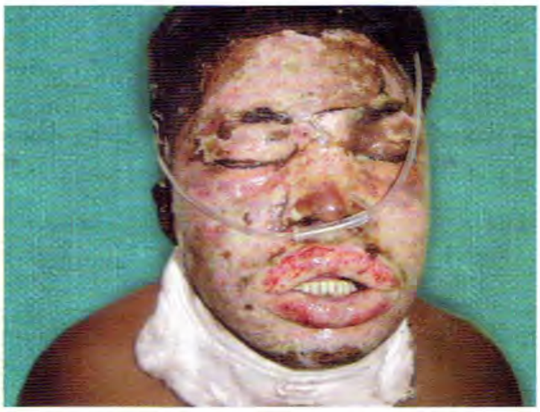

Fig. 11.5B: Same patient after 4 days of mechanical ventil Edema much reduced and patient extubated and then kept on prongs only

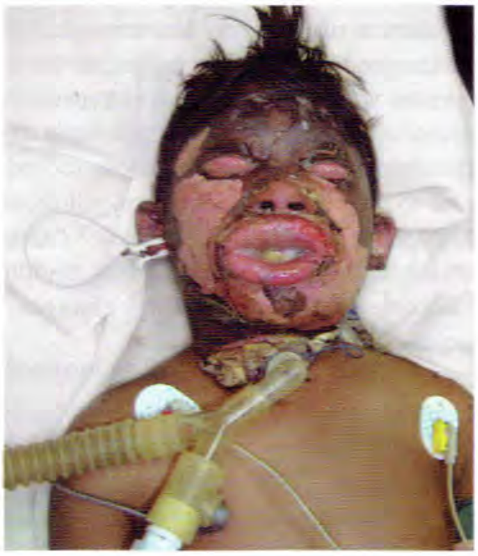

Fig. 11.6A: Severe facial burns with inhalation injury—Tracheostomy was done for ventilation

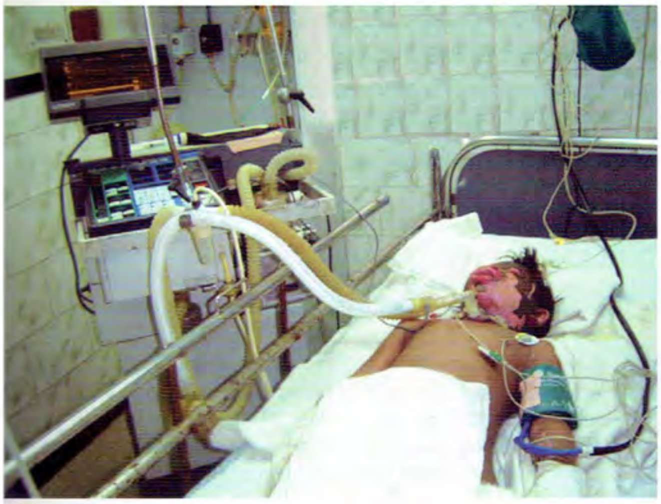

Fig. 11.6B: Same patient on ventilator

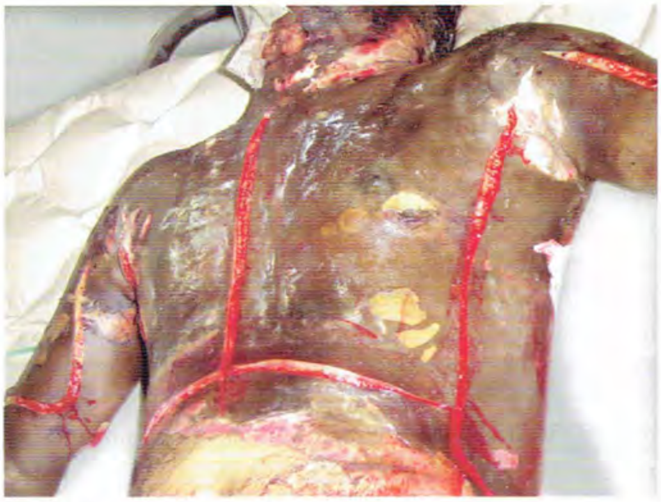

Fig. 11.8: Escharotomy incisions on chest in case of full thickness circumferential chest burns

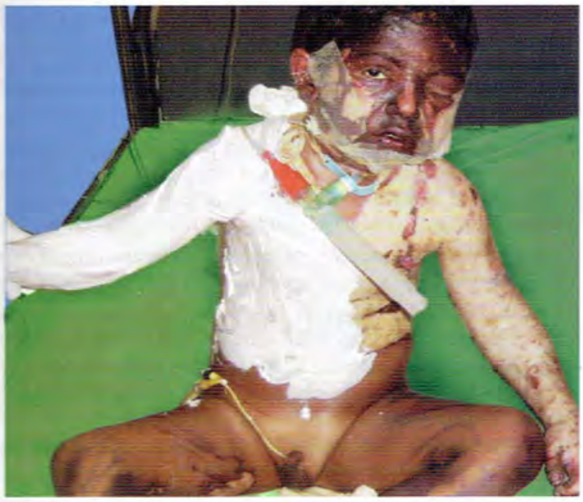

Fig. 11.7: Patient with tracheostomy, improved after mechanical ventilation and is being weaned off gradually

Preventive measures include meticulous wound care, pulmonary toilet, early removal of central lines and early extubation. Susceptible individuals (inhalation injury plus > 20% BSA burn) should undergo the following additional measures: daily WBC count, X-ray chest, and Gram's stain of sputum, sputum culture (every 3 days), early and effective treatment of burn wound (excision and grafting) etc.

Pneumonia usually develops during the second week after burn. It is evidenced by fever, sepsis, leucocytosis and abnormal X-ray chest. Antibiotic use should be according to microbiological data. Pulmonary toilet and possible ventilatory support may be required. Development of ARDS is a sign of poor prognosis.

Basic therapeutic priorities in patients with inhalation injury are as follows (Table 11.4):

Table 11.4: Basic therapeutic priorities in patients with inhalation injury

Respiratory problem	*Response (s)*
Asphyxia	Oxygenation, antidotes
Upper airway obstruction	Early, elective endotracheal intubation (rarely, tracheostomy)
Lower airway obstruction	
Mild	**Pulmonary toilet** **Bronchodilators** **Occasional therapeutic bronchoscopy**
Severe (hypoventilatory failure)	**Mechanical ventilation**
Parenchymal injury	
Mild	Supplemental oxygen (e.g. Low FiO_2 achievable by nasal prongs/mask)
Severe (with hypoxemic respiratory failure)	*Oxygenate (High FiO_2 [O_2 via ET tube])* Increase lung volume (CPAP, PEEP) Minimize ongoing leak adjustments of resuscitative fluid volumes, composition, and infusion rates.

Asphyxia is managed with oxygenation and specific antidotes; upper airway obstruction with early, elective endotracheal intubation (rarely, tracheostomy). Mild lower airway obstruction is managed with pulmonary toilet, bronchodilators and occasional therapeutic bronchoscopy. In case of severe ventilatory failure, mechanical ventilation is instituted (Fig. 11.6). Mild parenchymal injury is managed with supplemental oxygen (e.g. low FiO_2 achievable by nasal prongs/mask) and severe parenchymal injury (with hypoxemic respiratory failure) is managed with oxygen (high FiO_2 [O_2 via endotracheal tube]), increase in lung volume (CPAP, PEEP) and minimizing ongoing leak, adjustments of resuscitative fluid volumes, composition and infusion rates.

SUMMARY OF ACUTE HOSPITAL MANAGEMENT

The salient points that need to be emphasized in the acute care of patients presenting with inhalation injury are as follows:

1. Circumstances of the inhalation, e.g. history of a suicide attempt warrants careful patient observation and suggests early involvement of psychiatric personnel.
2. The accident scene – Inhalation injuries sustained in "closed spaces" (e.g., buildings, automobiles) result in severe respiratory abnormalities with greater frequency than those sustained in "open space".
3. Duration of exposure – An estimation of the severity of inhalation injury can be made with this information, as shown in animal studies.
4. Types of smoke involved (i.e. type of substrate burned): Very severe parenchymal lung injuries have been reported after exposure to polyvinylchloride smoke (plastics, plumbing, electrical insulation), polyurethane foam (furniture padding) smoke exposures, thus raising the index of suspicion for lower respiratory tract injury.
5. Time between initial resuscitation and hospital arrival – Together with knowledge of CO elimination half-life (largely based on estimates of the inspired O_2 fraction) and measured blood carboxyhemoglobin (COHb) level allows an estimate of peak COHb level. COHb levels have been shown to correlate with inhalation injury.
6. Patient's past medical history – The presence of underlying bronchospastic lung disease, for example, may alter subsequent therapy.

The long-term prognosis of patients with inhalation injury is variable. Degree of recovery depends on the extent of pulmonary parenchymal injury and subsequent hypoxic damage to the organs. Residual reactive airway diseas bronchiectasis, bronchiolitis obliterans and interstitial fibros may persist. Airway hyper-reactivity generally improves ov several months following inhalation injury. Cough, whee and shortness of breath may, however, persist even aft mild inhalation injury.

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Chapter

12

Fluid, Electrolyte and Acid-base Disturbances in Burns

Sujata Sarabahi

Electrolyte and acid-base disturbances can occur in a burn patient from immediately post burn till any time during the convalescent period and the severity of these disturbances are directly related to the initial resuscitation therapy given to the patient. Timely resuscitation has reduced the incidence of oliguria leading to acute renal failure even in the most extensive burns and thus, hypovolemic shock is not the most common cause of death in burns any longer.

HYPOVOLEMIA AND OLIGURIA

Hypovolemia following burns usually responds to increasing fluid therapy. However, hypovolemia leading to oliguria may be seen in two instances following burns, namely a) High voltage electrical injury or associated crush injury leading to myoglobinuria which blocks renal tubules and b) Extensive burns patient whose initial resuscitation has been delayed. In these cases, the oliguria does not respond to fluid load alone and require addition of an osmotic diuretic (mannitol).

An osmotic diuretic is indicated for electrical injury for flushing the renal tubules and avoiding precipitation of hemochromogens in the tubules. An initial loading dose of 25 gm of mannitol followed by 12.5 gm of mannitol with each 1 liter of fluid is given till urinary output is adequate. After administering a diuretic, the urinary output can no longer be used as an indicator for adequacy of fluid resuscitation.

Apart from the initial fluid loss following burns which is easily correctable, there is a continuous loss of watery exudates from the burnt surface even in the convalescent period which needs to be replaced in order to prevent dehydration. The exact volume of fluid lost from the wounds which is to be replaced is difficult to estimate. By and large, in burns treated by exposure method at normal room temperature, the daily evaporative and respiratory losses are calculated as 0.3 ml/cm^2 of body surface area burned (Davies, Lawke and Lilejedahl, (1974). The total body surface area is calculated from the formula of Du Bois and Du Bois, 1916 as follows:

Area in cm ^{2} = W $^{0.425}$ × H $^{0.725}$ × 71.84

(W= weight in kg and H= height in cm)

(The Du Bois nomogram is given in the appendix for easy calculations.)

Another formula given by Scott McDougall et al (1978) is as follows:

Evaporative water loss in ml/hour is = (25+ % BSA burn) × Total Body Surface Area in m^2.

These formulae are only very general guidelines and depend on various factors. When using them to estimate daily fluid requirements, one has to consider the following factors as well: whether patient is having high temperature or tachypnea, the environmental temperature (controlled and optimum or uncontrolled and same as outside), urinary output and whether closed or exposed technique for dressing wounds is used. Practically, if the wounds are dressed, the normal daily requirement (an average of 2.5-3.0 liters/day for an adult) is added to the amount calculated by these formulae. Last but not the least, monitoring of the burn patient, clinical and by laboratory investigations, is essential for individual modifications.

- Promote urinary potassium loss by loop diuretics.
- Potassium exchange resins.
- Hemodialysis.

HYPOCALCEMIA

Mild hypocalcemia (6-7 mg/dl) due to hypoalbuminemia and calcium sequestration in burned tissue may be seen in moderate burns (> 30%TBSA). However, this may not manifest clinically. Depression of serum albumin by 1g/dl lowers the total serum calcium concentration by approximately 1mg/dl, though the ionized calcium is usually normal. Marked alkalosis also lowers the serum calcium by increasing the protein binding. Severe hypocalcemia may result from either alkalosis in shock stage or following use of 0.5% silver nitrate soaks which may manifest clinically with cramps, facial nerve twitching, carpopedal spasms, tetany, Chvostek's sign, etc. This requires correction by intravenous calcium gluconate.

There is evidence that calcium administration in the critically ill may cause hypoxic cell damage, so calcium should be corrected in this setting only if the patient is symptomatic. Ionized calcium also should be determined to ascertain whether or not true hypocalcemia exists. When indicated, treatment of symptomatic hypocalcemia consists of 100-200 mg of elemental calcium (as 10% calcium gluconate) intravenously followed by a slow infusion of 0.5-2.0 mg/kg /hour with frequent determination of total and ionized calcium levels as well as correction of underlying cause. It is crucial to ensure that hypomagnesemia is corrected as this may potentiate the tendency to tetani and arrhythmias.

HYPERCALCEMIA

It is a rare problem in burn patients.

HYPOMAGNESEMIA

It is rarely seen in burn patients if adequate nutrition is ensured. If the patient is on prolonged Total Parenteral Nutrition (TPN) with inadequate magnesium supplements or if there is excess gastrointestinal and wound fluid losses, hypomagnesemia may manifest clinically with neurological signs, disorientation, neuromuscular irritability, seizures and tetany. This can be corrected by intravenous magnesium chloride or magnesium sulfate. Magnesium is best retained when given slowly over several hours.

HYPOPHOSPHATEMIA

Hypophosphatemia with serum phosphorus levels less t
1 mg/dl is observed in extensive burns that require la
amount of fluid for resuscitation leading to dilutional eff
and also receiving large volumes of intravenous gluc
solutions and antacid therapy. Therefore, it may be safer
monitor serum phosphorus levels as well. Phosphorus pl
an important role in oxygen unloading from red cel
muscular activity and neutrophil function. Ente
replacement of phosphorus is usually sufficient, althoug
levels are really low, intravenous sodium or potassi
phosphate (0.08-0.16 mmol/kg of sodium phosphate in 5
ml 0.45 normal saline over 6 hours) may be administe
with caution only if phosphate is below 1.0 mg/dl.

ACID-BASE BALANCE

A burn patient may have metabolic or respiratory acido
or metabolic alkalosis.

Metabolic Acidosis

It is usually seen in early phase of extensive burns or
case the patient develops sick cell syndrome. It may e
be seen on admission in pediatric age group in less tha
years of age since their buffering capacity is less. Oligu
due to insufficient or delayed fluid resuscitation may elev
blood lactate levels to 2 mEq/L or more due to anaero
metabolism. This condition is diagnosed by low arterial
in combination with low arterial PCO_2. It can be correc
by increasing fluid infusion and oxygen therapy
hypoxemia. If blood pH falls to below 7.1, it should
corrected by sodabicarb intravenously. However, in gene
the underlying cause of the acidosis should be determi
and appropriate treatment undertaken. Empirical bicarbon
therapy can be complicated by intracellular acidosis. T
formula used for calculating the dose of sodabicarb is
follows:

Base deficit × 0.3 × body weight (in kg) = 8.4% NaHC
(in ml)

Half of this dose is given immediately and one h
after this correction, ABG is repeated and further correct
is done according to the new pH value in order to keep
pH above 7.1.

It has been noticed that if plasma is used for treat
burn shock, no early acidosis is seen because of alkalizat

produced by the citrate in the transfused plasma. If saline or dextran alone is used for resuscitation, the acidosis may occur and may require addition of bicarbonate. It is essential to check this early metabolic acidosis as it predisposes to acute renal failure.

Respiratory Acidosis

It is rarely seen in initial post burn period except in patients who have inhalational Injury which leads to pulmonary insufficiency or pulmonary edema or atelactasis, bronchopneumonia, pulmonary thromboembolism with or without sepsis. It is characterized by low pH, high pCO_2 and hypoxemia. The treatment includes attempt to identify and correct the underlying cause of the respiratory acidosis and in very severe cases by intubation and mechanical ventilation and antibiotics for treating sepsis.

Metabolic Alkalosis

It can occur following extensive burns and is attributed to the increased adrenocortical activity and also in response to respiratory alkalosis due to high excretion of catecholamines. It also follows prolonged hypokalemia, hyperaldosteronism and prolonged nasogastric suction due to paralytic ileus. This usually requires correction of hypokalemia and fluid replacement. Treatment consists of removal of the precipitating factors and correction of electrolyte deficits to restore acid-base balance. Adequate chloride must be provided in those cases associated with volume and chloride deficit. Continued diuresis in the face of a metabolic alkalosis can be helped by acetazolamide which is a carbonic anhydrase inhibitor with potassium supplement to increase urinary bicarbonate excretion. Infact, acetazolamide is frequently used in chloride resistant metabolic alkalosis.

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Chapter 13

Acute Renal Failure in Burns and its Management

Rajiv Kohli, Arun Goe

The initial effects of a burn injury on kidney and its functions are in response to circulatory and metabolic consequences followed later by effects of treatment and complications. After an extensive burn injury, there occurs a marked shift of intravascular fluid into the interstitial spaces all over the body including the non-burnt sites resulting in hypovolemia and decreased circulation. This in turn, decreases renal perfusion and urine output. Urinary specific gravity and osmolality increases. With timely and appropriate fluid and electrolyte replacement therapy, the hypovolemia is corrected and normal circulation is restored. Once the capillary permeability gets restored, the fluid is withdrawn from the interstitial spaces back into the intravascular compartment and diuresis occurs to remove this excess fluid. Additionally, the renal function is also altered as a direct effect of the stress of burn trauma in response to which the posterior pituitary releases antidiuretic hormone (ADH) and maximum reabsorption of water occurs. In addition to ADH, aldosterone is released from adrenal cortex and maximum sodium is absorbed. Thus, under the influence of ADH and aldosterone, the kidney excretes an amount of urine just sufficient to handle the normal solute load, largely represented by urea.

If the resuscitation is delayed and/or inadequate, renal blood flow and glomerular filtration rate decrease and may ultimately lead to prerenal form of acute renal failure. This may progress to intrinsic renal failure which is usually caused by septicemia and nephrotoxic drugs. Renal failure may become part of multi-organ dysfunction syndrome (MODS). In extensive thermal burns, damage to red cells and in contact electrical burns, necrosis of muscles may release hemoglobin and myoglobin pigments respectively into circulation leading to blockage of renal tubules, again leading to renal failure.

Acute renal failure (ARF) or Acute kidney injury (AKI) as it is known these days, is one of the most devastating complication of burns with a high mortality rate. It has been said to affect from 0.5 to 30% of the patients admitted to a burn unit. The incidence depends not only on the severity of burn injury but also on the definition of acute renal failure used. Once the acute renal failure develops in a burn patient, the prognosis becomes remarkably unfavorable. This fact has been recognized for over 50 years now and most studies have reported a mortality rate of around 80% in those patients who require dialytic support. However, the prognostic importance of less severe forms of acute kidney injury, not requiring dialysis, is not well documented.

ETIOLOGICAL FACTORS IN ACUTE RENAL FAILURE IN BURNS

Depending upon the interval between the time of burn injury and the onset of acute renal failure, two different forms of ARF have been described in burn patients. Burns affecting more than 20% of skin surface are likely to cause enough loss of extracellular fluid and albumin from plasma volume to produce shock and hypoalbuminemia. The ARF occurring in the first few days (within 5 days) after thermal injury is related to a decrease in renal perfusion resulting from hypovolemia (due to fluid shifts from the intravascular compartment into the interstitial space and extensive fluid losses from the burnt areas which may get accentuated by a delay in fluid resuscitation), low cardiac output and systemic vasoconstriction during the resuscitation period or to myoglobinuria with damage to tubular cells. Elevated levels of stress related hormones, such as catecholamines, angiotensin, aldosterone and vasopressin have been implicated in the pathogenesis of this form of ARF. This form of ARF occurs less frequently than before because of the widespread availability of cheap intravenous electrolyte solutions and the aggressive fluid resuscitation performed nowadays in most burn units. However, this is still a life threatening complication in patients with extensive deep burns and those with electrical burns.

The other form of ARF developing at a later date (after 5 days) has more complex pathogenesis. This form has been reported to be related to the sepsis and multi-organ dysfunction syndrome (MODS) and very often proves fatal. It occurs more commonly in patients with inhalational injury and is presently the most frequent cause of renal insufficiency in burned patients. Administration of nephrotoxic drugs like aminoglycosides, vancomycin and amphotericin B etc. are also contributing factors in the development of ARF.

Various studies done have shown significant correlation between total body surface area burnt and presence of inhalation injury and development of ARF. Parameters like age, third degree burns or electrical injury were not significant.

DEFINING, QUANTIFYING AND CLASSIFYING ACUTE RENAL FAILURE

Acute renal failure is characterized by an abrupt and sustained fall in glomerular filtration rate (GFR) leading to accumulation of nitrogenous (clinically manifesting as a rise in urea and creatinine) and non-nitrogenous waste products. This potentially reversible condition is accompanied by metabolic disturbances such as metabolic acidosis and hyperkalemia, changes in the body fluid balance and effects on many other organ systems.

The two major functions of a healthy kidney are excretion of nitrogenous wastes and formation of urine. Creatinine is more specific in assessing renal function than blood urea nitrogen (BUN), but it corresponds only loosely to GFR. Same value of serum creatinine may correspond to different GFRs in two individuals with different body masses. However, rise in serum creatinine values corresponds to similar decline in GFR.

There is no universal definition of ARF. Common criteria are either biochemical (rise in serum creatinine of 0.5 mg/dl or 50% from baseline) or clinical (oliguria, urine output < 400 ml/day) or a combination of both. The acute dialysis quality_initiative (ADQI) group proposed the RIFLE SYSTEM classifying acute renal failure into 3 severity categories (**R**isk of renal dysfunction, **I**njury to the kidney and **F**ailure of renal function) and 2 clinical outcome categories (**L**oss of renal function and **E**nd stage renal disease) (Table 13.1). Loss (Persistent AKI) denotes the need for renal replacement therapy for more than 4 weeks whereas in ESRD, the need is for longer than 3 months.

CAUSES OF ACUTE RENAL FAILURE

The causes of ARF can be broadly grouped into three major categories. These are decreased renal blood flow (Prerenal causes), direct renal parenchymal damage (Intrinsic renal causes) and obstructed urine flow (Post renal or Obstructed cause). In burned patients, it is mainly the prerenal and the intrinsic renal failure that occur unless a patient of renal calculi gets burnt to have an additional postobstructive failure.

Prerenal Failure

In the prerenal form, there is a reversible increase in serum creatinine and BUN. It results from decreased renal perfusion, which leads to reduction in GFR. The main cause in burned patients is acute hypovolemia and hypotension.

Table 13.1: RIFLE classification (*Adapted from Bellomo et al, 2004*)

	GFR Criteria	*Urine Output Criteria*
Risk	Serum creatinine increased 1.5 times	< 0.5 ml/kg body weight/hour for 6 hours
Injury	Serum creatinine increased 2 times	< 0.5 ml/kg body weight/hour for 12 hours
Failure	Serum creatinine increased 3 times or creatinine = 4 mg%, when there was an acute rise of > 0.5 mg	< 0.3ml/kg body weight/hour for 24 hours or anuria for 12 hours
Loss	Persistent acute renal failure, complete loss of kidney function for longer than 4 weeks	
End stage renal disease(ESRD)	End stage renal disease for longer than 3 months	

This activates a number of neurohumoral vasoconstrictive systems to maintain them. Changes in preglomerular and postglomerular arteriolar resistance, mediated by prostaglandins and nitric oxide and activation of angiotensin II, enables renal blood flow and glomerular filtration rate to remain roughly constant across a wide range of mean arterial pressures. However, below a mean arterial pressure of 70 mmHg, this autoregulation is impaired and glomerular filtration rate falls proportionately. Administration of drugs that interfere with autoregulation, like non-steroidal anti-inflammatory drugs (NSAIDs) and ACE inhibitors, precipitate the prerenal failure.

Intrinsic Renal Failure

The cause of intrinsic renal failure is acute tubular necrosis (ATN), resulting from continuation of the same pathophysiological processes that lead to prerenal hypoperfusion (ischemic ATN). Drugs may also cause a direct damage to the renal tubules (nephrotoxic ATN). Intrinsic renal failure is often multifactorial. The most common cause in burned patients is sepsis and use of nephrotoxic drugs may also be a contributing factor. It is often accompanied by multi-organ failure.

DIAGNOSTIC APPROACH TO RENAL FAILURE

Serum Creatinine

In acute renal failure, renal function is commonly monitored by daily estimations of serum creatinine. However, it has limitations as a marker of GFR in patients with acute renal failure because its concentration depends not only on urinary clearance of creatinine but also on the rate of production and the volume of distribution. Serum creatinine levels do not reflect the GFR accurately in the non-steady-state condition of ARF.

Serum Cystatin C

Serum cystatin C, excreted by the glomerulus, is one of the newer markers. It is a cysteine proteinase inhibitor having a low molecular weight and is produced by nucleated cells independent of their pathologic status. Although some studies have found it to be an early and reliable marker, it has not yet been well validated as an indicator of GFR in acute renal failure as standardised assays are not available.

Urine Volume

Acute anuria or severe oliguria are quite specific clinical indicators of acute renal failure. While prerenal forms of ARF nearly always present with oliguria (< 400 ml/day in an adult), the renal and postrenal forms can be associated with any amount of urinary output from almost nil to normal to polyuria. A severe ARF can coexist with a normal urinary output. The change in the amount of urine excreted precedes the biochemical alterations.

Urinary Indices

If the renal tubular function is intact, renal vasoconstriction during the initiation phase of acute tubular necrosis is associated with increased tubular reabsorption and the fractional excretion of sodium (FE Na), i.e.

$$\frac{\text{Urinary sodium} \times \text{plasma creatinine}}{\text{Plasma sodium} \times \text{urinary creatinine}} \times 100$$

should be less than 1%. This physiological response is altered when the patient is administered diuretics in the form of furosemide or mannitol or has glucosuria. To differentiate between prerenal and renal forms of ARF, especially when the patient has received a diuretic, fractional excretion of urea is more specific and sensitive than that of sodium. Other urinary indices are shown in Table 13.2.

Biomarkers

A number of biomarkers have been proposed and are being studied for the early diagnosis of acute renal failure, for example, urinary interleukin 18 (IL-18) and tubular enzymes such as intestinal form of alkaline phosphatase, N acetyl-β glucosaminidase and alanine amino peptidase etc. However, more validation is required before their routine use.

PREVENTION OF ACUTE RENAL FAILURE

As is true for the burn injury itself, prevention of acute kidney injury is the desired goal of every clinician. There is clear evidence that a burn complicated by renal failure has a greater overall mortality. While prevention is in every clinician's domain, once the failure sets in, a nephrologist must be called upon by the burn surgeon for appropriate and specialized management. Various pharmacological and non-pharmacological strategies have been devised and used apart from some novel therapies.

Non-pharmacological Strategies

Fluids

An early and adequate fluid therapy is crucial in maintaining renal function in extensively burnt patients. Even a general practitioner who sees the patient first should be able to start an intravenous drip and infuse fluids before the patient is

Table 13.2: The most important urinary variables in the differential diagnosis between prerenal and renal acute renal failure (*Adapted and modified from:* Lameire et al, 2005)

Urine analysis	*Prerenal*	*Renal*
Specific gravity of urine	>1020	1010 approx.
Osmolality(mOsm/kg H_2O) of urine	>500	<350
Urinary sodium concentration (mmol/l)	<20	>40
Fractional excretion of sodium %	<1	>2
Fractional excretion of urea %	<35	>35
Fractional excretion of uric acid %	<7	>15
Fractional excretion of lithium %	<7	>20
Low molecular weight proteins	Low	High
Brush border enzymes	Low	High
Ratio of urinary to plasma creatinine	>40	<20
Ratio of urinary to plasma osmolality	>1.5	<1.1
Urinary sediment	Hyaline and fine granular casts	Coarsely granular and cellular casts

transported to an appropriate burn care facility. Intravascular volume correction is the single most important factor in preventing renal failure. With the widespread availability of cheap intravenous fluids, the incidence of renal failure in burns due to hypovolemic causes has certainly come down over the years. Various fluid formulae, such as Parkland or Brooks, are just general guidelines. Which is the optimal resuscitation fluid is still debated. Normal saline and 4 % albumin have clinical equivalence, shown by SAFE study of patients admitted in ICU'S in Australia and New Zealand. Once the patient is considered euvolemic (normotensive, no postural drop, JVP and/or CVP normal), care should be taken to avoid fluid overload and a maintenance regime started, which takes account of renal and insensible losses accounting for a positive balance of 500 ml/day. Hourly input should equal previous hour's output plus 25 ml. This requires accurate observation and record keeping by the nursing staff and reassessment by the doctors.

Nephrotoxin Exposure

Minimal nephrotoxin exposure is an important strategy to prevent ARF. Antibiotics like aminoglycosides, amphotericin B and vancomycin, etc. should be used carefully with regular monitoring of renal function and if possible, serum levels of these drugs should also be monitored. It is important to avoid drugs like NSAIDs and ACE inhibitors during pre-renal phase of ARF.

Pharmacological Strategies for Prevention of Acute Renal Failure

Dopamine

The use of low dose (1-3 μgm/kg/minute) dopamine has been advocated to increase renal perfusion in critically ill patients. Recent studies including a large randomized controlled trial have shown it to lack efficacy on renal outcome or overall mortality. Use of dopamine may also reduce splanchnic perfusion, depress respiration, suppress anterior pituitary hormone release and function and worsen renal function in hypovolemic or normovolemic patients. Its routine use in ARF is not currently justified.

Diuretics

There is a theoretical rationale for the use of loop diuretics in ARF – inhibition of Na^+/ K^+/Cl^- pump in the thick ascending limb of loop of Henle, with subsequent decrease in Na^+/K^+—ATPase activity, should reduce the oxygen requirement of these cells and thus, their susceptibility to ischemic damage. However, there are scarce clinical data to support this and recent studies have either correlated the use of diuretics with increased mortality or shown no benefit. It seems reasonable to use diuretics only in adequately resuscitated oliguric patients at a dose suitable to the degree of renal impairment (250 mg furosemide intravenous over one hour is a standard regimen) and to stop diuretics if oliguria persists. Converting oliguric to non-oliguric renal failure may help with fluid and electrolyte management but does not seem to affect eventual need for dialysis or overall mortality and should not delay the start of renal replacement therapy.

Other Therapeutic Measures and Novel Therapies

Various other agents have been studied for potential use in prevention of acute tubular necrosis, e.g. Fenoldopam, atrial natriuretic peptide (ANP), theophylline, N-acetylcysteine, calcium-channel blockers, epidermal and insulin-like growth factors, scavengers of oxygen free radicals and prostaglandins, erythropoietin, etc. None of these novel therapies have been proven to be effective or safe in human acute renal failure.

MANAGEMENT OF ACUTE RENAL FAILURE

When a burn patient goes into acute renal failure, the management is based on supportive care as there is no definitive pharmacological treatment. Once the supportive treatment fails, initiation of renal replacement therapy is desired before uremic complications emerge.

Supportive Treatment of ARF

The aim of treatment of ARF in burns is to prevent further fall in renal function. Supportive treatment mainly consists of maintaining metabolism, taking care of life threatening complications like hyperkalemia, pulmonary edema, metabolic acidosis and taking care of nutrition of the patient and treatment of infections.

Fluid Balance

An accurate fluid balance should be maintained by daily measurements of body weight and Input/Output. In oliguric patients, daily fluid intake should be limited to 500 ml plus the previous day's urinary output unless there are signs of volume depletion/overload. Dietary sodium is restricted to 2 gm/day. In the absence of hyperalimentation, an individual with acute tubular necrosis is expected to lose 0.3-0.5 kg body weight daily. If this expected weight loss does not occur or there is weight gain, fluid therapy must be reassessed. Hyponatremia might necessitate stringent free water restriction.

Acidosis

Severe metabolic acidosis ($pH < 7.2$) often occurs once ARF develops and arises through a variety of mechanisms related both to reduced renal function and the underlying cause of illness. Systemic acidosis impairs cardiac contractility, induces bradycardia, produces vasodilatation and augments hyperkalemia. Reversing acidosis through administration of sodium bicarbonate solution seems sensible although there is very little evidence to show that it provides benefit. It can cause volume overload by increasing the sodium load. Hemodialysis or hemofiltration is usually required to treat severe acidosis in oligoanuric patients.

Hyperkalemia

Hyperkalemia is defined as serum potassium levels of more than 5.5 mmol/l. Severe hyperkalemia (i.e., plasma potassium > 6.5 mmol/l) is a medical emergency because of risk of cardiac arrhythmias. Emergency treatment of hyperkalemia primarily dictated by changes in ECG includes the use of intravenous calcium, driving potassium into the cells with glucose and insulin or nebulization with salbutamol. If these measures fail, emergency dialysis is indicated to save the patient's life. The treatment of hyperkalemia is summarized in Table 13.3.

Pulmonary Edema

Oligoanuric patient with pulmonary edema resulting from fluid overload represents a clinical challenge. If significant ventilatory failure is present, supplementary oxygen, non-invasive ventilation or intubation and ventilation are to be started. Pharmacological treatment to offload the decompensated heart can be started in the form of intravenous opioids (diamorphine 2.5mg) and intravenous infusion of nitrate (glyceryl trinitrate 50 mg in 50 ml of 0.9% saline at a rate of 2- 20 ml/hour, keeping the systolic blood pressure >95 mm Hg) and attempts to provoke diuresis by furosemide 250 mg in 100 ml saline over 1 hour. If these measures fail, immediate hemodialysis or hemofiltration is to be done.

Table 13.3: Summary of treatment strategies in hyperkalemia

Treatment	*Immediacy of action*	*Reduction in K^+*	*Duration of action*
10% calcium gluconate –10-20 ml I/V over 2-5 min	1 -3 minutes	Nil	30-60 min
Insulin/glucose 10 units plain insulin in 50 ml of 50% dextrose infused over 10-20 min	15 -30 minutes	0.65 -1.0 mmol/l	4-6 hours
Salbutamol (nebulized/intravenous) 10-20 mg via nebulizer or 0.5 mg I/V	30 minutes	0.6-1.0 mmol/l	2-4 hours
Ion exchange resin 15 gm three times a day orally	2-3 hours	0.5 -1.0 mmol/gm resin	4-6 hours
Hemodialysis	immediate	< 1.5 mmol/l	when dialysis is ongoing

Nutritional Support

The goal of nutritional support is to preserve lean body mass, maintain immunocompetence, offer substrate for the acute inflammatory state and help promote wound healing. It is an important part of the treatment of the burnt patient as a whole, with or without renal failure. As these patients are in a hypercatabolic state due to both burn as well as ARF, they are to be given hypercaloric diet. This intake must be without excessive volume. Carbohydrate intake should be sufficient (> 100 gm/day) to avoid breakdown of endogenous proteins for glucose. Combination of carbohydrates and lipids should be used. These patients also require enough protein of high quality. As far as possible, enteral feeding should be preferred over parenteral feeds. Fulfilling the nutritional requirement may not be possible without the dialytic support.

Renal Replacement Therapy

In the last 15 years a number of advances have been made in the renal replacement therapy in the management of acute renal failure. However, as there are several techniques available, there is controversy about selection of the best modality for a given patient. Also, there are no absolute rules as to when renal replacement therapy should begin but too soon is better than too late and the treatment should be started before complications occur. In general, the indications for dialysis fall into three broad categories, viz., solute indications, e.g. marked azotemia, volume indications, e.g. fluid overload, or both.

The following are the criteria for starting renal replacement therapy:

1. Oliguria, with urine output less than 200 ml/12 hours.
2. Anuria, with urine output less than 50 ml/12hours.
3. Hyperkalemia, serum potassium > 6.5 mmol/l
4. Severe acidosis, pH < 7
5. Azotemia, blood urea > 200 mg/dl
6. Complication of severe uremia viz., encephalopathy, pericarditis, neuropathy/myopathy, etc.
7. Plasma sodium > 155 mmol/l or < 120 mmol/l.

MODE OF RENAL REPLACEMENT THERAPY (DIALYSIS)

Dialysis is a process in which molecules from one solution (i.e., blood) diffuse across a semi-permeable membrane into another solution (i.e., dialysate). This transfer of solute across the membrane is determined by the characteristics of the membrane and the solute concentration on its the two sides. The movement of small molecular weight solutes from the blood to dialysate under the driving force of an electrochemical gradient is called diffusive clearance. Convective clearance (Ultrafiltration), on the other hand, occurs when water is driven across the membrane by hydrostatic/osmotic forces. Solutes that can pass through the membrane pores move along with water (solute drag).

Vascular Access

For hemodialysis, a good vascular access is required to withdraw the blood from the patient, to be passed through dialyser and then returned back. Effective vascular access can be in the form of placing one catheter in an artery and another in a vein. A double lumen catheter can be placed in a central vein like femoral, internal jugular or subclavian and blood can be withdrawn through one lumen and returned back through the other with the help of a pump.

Anticoagulation

To avoid clotting in a dialysis membrane or circuit tubing or at the vascular access, anticoagulants are generally required in patients undergoing dialysis. These are generally in the form of unfractionated heparin or low molecular weight heparins. Regional citrate anticoagulation or frequent saline flushing of the system is also used in patients in whom heparin cannot be used.

Types of Dialysis Modalities

Two types of dialysis modalities are available:

1. Intermittent therapy, an extracorporeal dialysis, where the patient is treated for less than 24 hours.
2. Continuous therapy [(Continuous Renal Replacement Therapy, (**CRRT**)], again an extracorporeal dialysis, where the patient is treated for 24 hours or more.

These modalities are detailed in Table 13.4.

Which Modality to be Used?

Both the modalities have their advantages and disadvantages as shown in Table 13.5. Various studies done have proved that both the modalities are equally effective in treating such patients. Mortality rates are also similar for both the modalities. The use of a particular modality depends upon the patient's condition, availability of infrastructure at a particular center, availability of trained manpower and experience of physicians and nurses in handling such patients.

Table 13.4: Dialysis modalities for acute renal failure (*Adapted and modified from:* Abdeen et al, 2002)

Intermittent therapies	*Continuous therapies (CRRT)*
Hemodialysis	Peritoneal dialysis
Hemofiltration	Ultrafiltration (SCUF)
Ultrafiltration	Hemofiltration (CAVH , CVVH)
Extended daily dialysis (EDD)	Hemodialysis (CAVHD ,CVVHD)
	hemodiafiltration (CAVHDF , CVVHDF)
Slow low efficiency dialysis(SLED)	

SCUF, Slow Continuous Ultra Filtration; CAVH, Continuous Arterio Venous Hemofiltration; CVVH, Continuous Veno Venous Hemofiltra CAVHD, Continuous Arterio Venous Hemo Dialysis; CVVHD, Continuous Veno Venous Hemo Dialysis; CAVHDF, Continuous ArterioVenous Dia Filtration; CVVHDF, Continuous VenoVenous Hemo Dia Filtration; SLED, Slow Low Efficiency Dialysis

Table 13.5: Advantages and Disadvantages of Intermittent versus continuous renal replacement therapy (*Adapted and modified from:* Fry et al, 2006)

	Intermittent hemodialysis	*Continuous renal replacement therapy*
ADVANTAGES	• Lower risk of systemic bleeding • More time available for diagnostic and therapeutic interventions • More suitable in severe hyperkalemia • Low cost	• Better hemodynamic stability • Fewer cardiac arrhythmias • Better pulmonary gas exchange • Better fluid control • Better biochemical control • Shorter stay in ICU • Good removal of inflammatory mediators • Can be done at bed side
DISADVANTAGES	• Availability of dialysis staff • More difficult hemodynamic control • Inadequate dialysis dose • Inadequate fluid control • Inadequate nutritional support • Poor removal of inflammatory mediators	• Greater vascular access problem • Higher risk of systemic bleeding • Long term immobilization of patient • More filter problem • Higher cost

Peritoneal dialysis may not be possible in burn patients as the area may be burnt and chances of infections are also more. Intermittent therapies may be useful if more solute is to be removed as in patients with sepsis. Intermittent therapy also leaves time for any surgical procedure(s) which might be required in such patients.

If fluid removal is a major concern, continuous therapies are more useful and a lot of fluid can be removed to help give the drugs and nutritional support to such patients. In hemodynamically unstable patients who have low blood pressure, continuous therapy is more useful. For burn patients CVVHD or CVVH is the therapy of choice in most of the patients.

Dose of Dialysis

It is how much dialysis has to be given to the patient. In End Stage Renal Disease (ESRD) the KT/V of 1.2 per treatment is the acceptable minimum dose. Here K represents urea clearance, T is the time of dialysis and V is the volume of urea distribution. Patients with ESRD have a relati constant urea generation rate and are in a steady st Patients of ARF tend to have a fluctuating body f composition and varying urea generation rates. Th formulae may not always be correct. Nonetheless th treatments per week with KT/V of 1.2 seems good eno for ARF patients. Some studies have suggested that d dialysis may be more useful for early recovery of th patients.

In CRRT, the dose of therapy is correlated with effl flow rate. There are studies comparing higher ultrafiltra with low ultrafiltration rates but the results are conflict There is some consensus that ultrafiltration rates of 35 kg/hour show better results.

THE OUTCOME AND PROGNOSIS

Outcome of burn patients with Acute Kidney Injury (A is poor. These patients require prolonged hospital s prolonged ICU treatment, greater need for ventilator prolonged stay on ventilators. Recent studies have shown that progressive renal injury, as suggested by RIFLE criteria, have poorer outcomes. Despite much progress in treatment modalities, mortality in a burn patient with ARF requiring renal replacement remains high and in various studies it is around 80% as shown in Table 13.6.

Table 13.6: Mortality rates in severely burned patients with acute renal failure requiring renal replacement therapy (*Adapted and modified from:* Holm et al, 2000)

Author	*No. of patients with ARF*	*Mortality (No. of patients)*
Cason (1953-1956)	49	100%(49)
Vertel et al (1960-1966)	24	88% (21)
Cameron et al (1967)	22	96% (21)
Davies et al (1958-1979)	28	86% (24)
Davies et al (1990-1991)	15	80% (12)
Leslanc et al (1987-1994)	16	82%(13)
Holm et al(1994-1998)	48	85%(41)
Kim et al (2000)	28	100%(28)
Coca et al (1998-2003)		73%

The prognosis of severely burned patients with ARF has not improved for the last 50 years. It may be because of the fact that the burn patients developing ARF generally have multi-organ failure resulting in high mortality.

SUMMARY AND CONCLUSION

Acute renal failure is fairly common in burn patients. Patients with high total body surface area (TBSA) burns have higher chances of developing acute renal failure (ARF). With better fluid resuscitation, majority of these patients develop acute renal failure later in the course of their illness due to sepsis and associated multi-organ failure. Management of ARF should include early diagnosis and renal replacement therapy, which ideally should be performed as a continuous veno-venous hemofiltration. The modern approach with aggressive fluid resuscitation which is now practiced in most burn centers should be continued as this seems to effectively prevent early prerenal failure.

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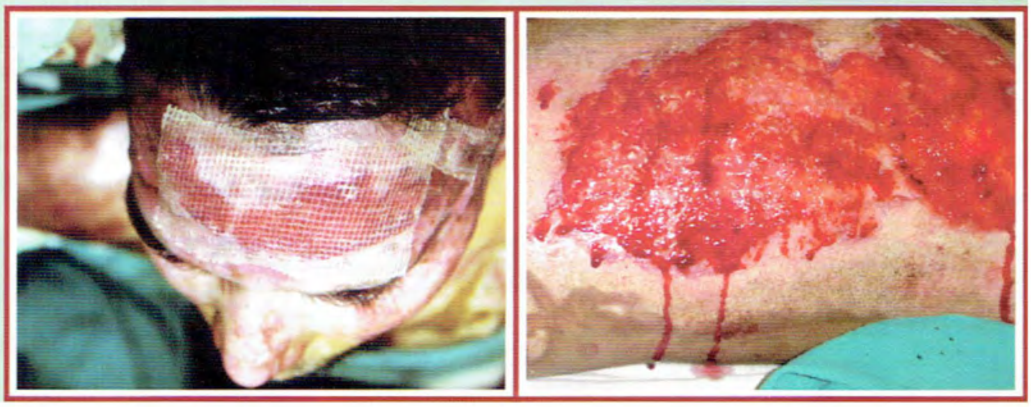
3

Section

Local Care of Burn Wound



Chapter 14

Burn Wound Management

Sujata Sarabahi

EVALUATION OF BURN WOUND

Introduction

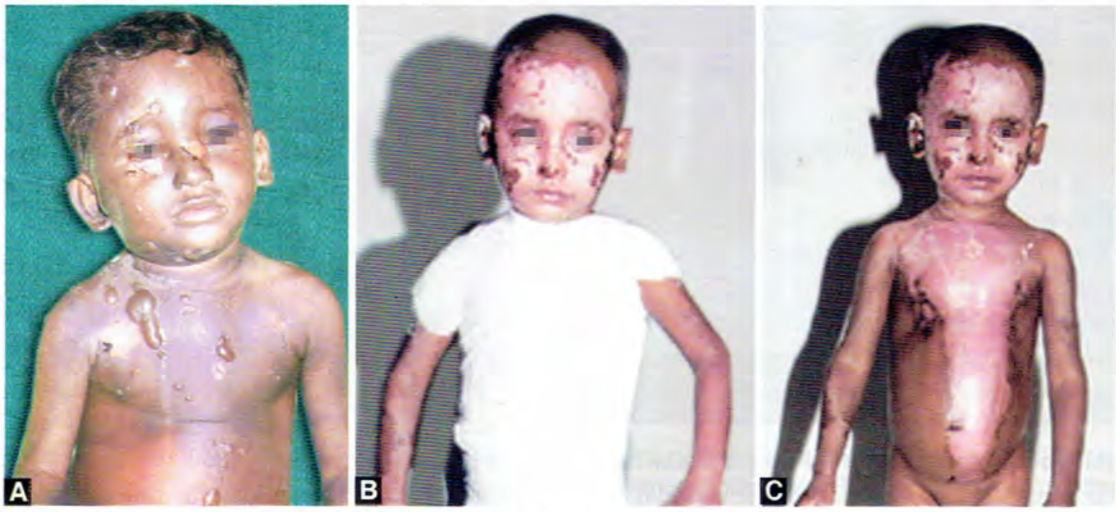
The survival of a moderate to extensively burnt patient depends on the successful management of the burn wound. A burn patient is always at risk as long as his/her wounds persist. There is a general consensus that burn wound infection is the primary cause of morbidity and mortality following an extensive burn injury. Hence, an early wound closure can eliminate not only the risk of infection but also the risk to life of the extensively burnt victim.

After the initial management and resuscitation of a burn patient is complete, the focus shifts to the management of the burn wound. This management requires a careful evaluation of the burn wound.

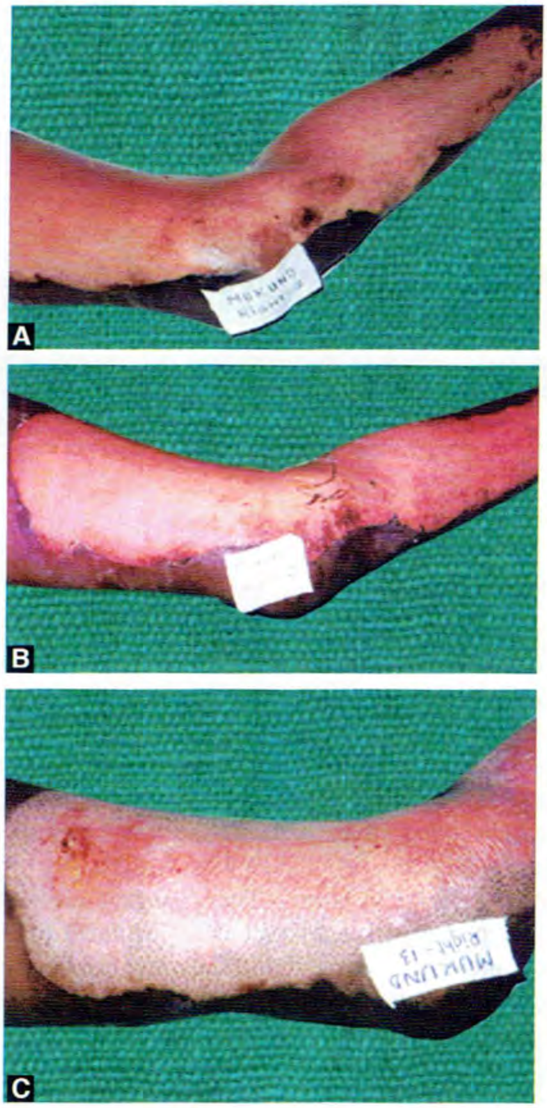
Wound Evaluation

A burn wound is evaluated with respect to its site, extent and depth in order to decide the appropriate method(s) of treatment. The methods for assessment of the extent and depth of burn wounds have been discussed in previous chapters elsewhere in detail in this volume. For practical purposes for patient management, the burn wounds can be categorized into 3 groups when first seen by a burn specialist in the casualty:

a. Partial thickness burns—They can heal on their own with conservative treatment in the form of topical antimicrobial treatment and dressings which facilitate spontaneous healing from the remaining skin elements over a period of upto 3 weeks (Figs 14.1A to 14.2C).
b. Full thickness burns –They require surgical intervention as there are no chances of spontaneous healing because



Figs 14.1A to C: (A) Superficial partial thickness burn in a child. (B) closed dressing. (C) Complete healing in 9 days



Figs 14.2A to C: Partial thickness burn, healing with conventional dressings by day 13 post burn

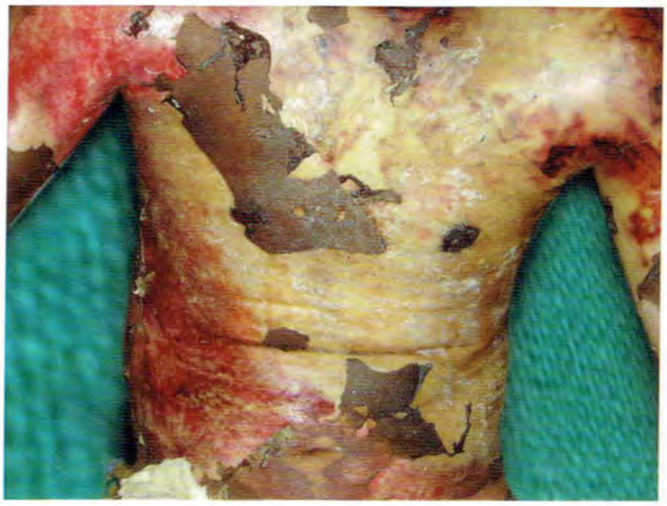


Fig. 14.3: Full thickness burns requiring surgical intervention

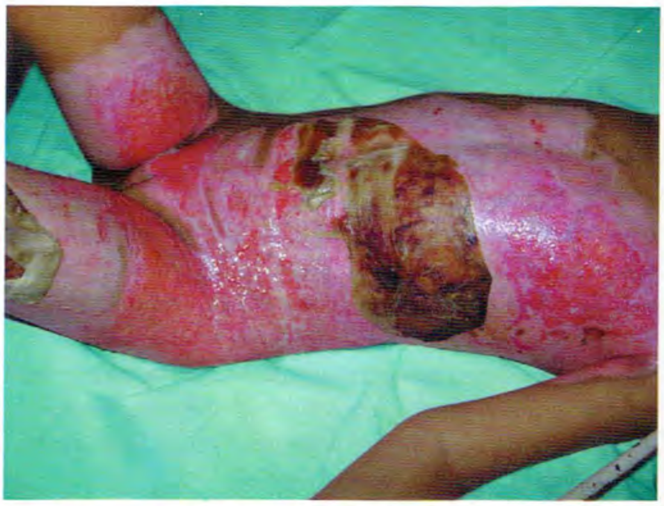


Fig. 14.4: Mixed burns in a child –Reassessment of depth done at 10 days

of loss of all dermal elements. The type of surgical intervention and timing of surgery eventually affect the outcome of these wounds (Fig. 14.3).

c. Indeterminate depth burns—They cannot be clearly allocated to either of above two groups in the **first look**. Their potential for regeneration is doubtful. These areas require initial conservative management followed by a **second look** after 10-14 days when they may reveal their true depth. A definitive plan is then formulated (Fig. 14.4).

Partial thickness burns involving dermis may be superficial dermal or deep dermal. The superficial dermal burns which involve the papillary layer of dermis are typically pink and moist (Fig. 14.5), whereas, deep dermal burns involve the reticular layer and are dry and pearly (waxy) white in color (Fig. 14.6A). Full thickness burns (Fig. 14.6B), as the name indicates, involve the whole of epidermis and dermis and appear parchment like with visible, thrombosed subcutaneous veins. As the sensory end organs are concentrated in the skin, the presence of sensation (tested by pinprick) in the burnt area indicates that the skin is viable and the burn is partial thickness whereas absence of sensation indicates full thickness burns. Similarly, easy removal of hair (by pulling) indicates deeper burns whereas

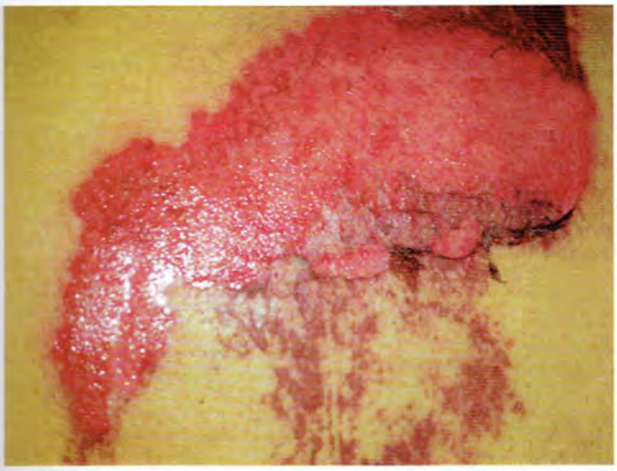


Fig. 14.5: Superficial dermal or superficial partial thickness burns with moist and red look

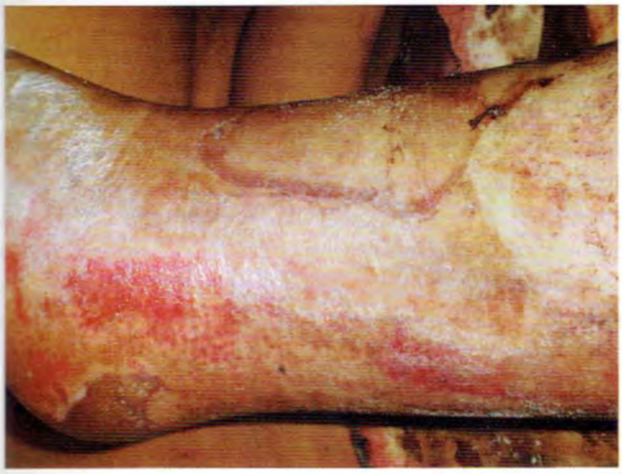


Fig. 14.6A: Deep dermal burns with pearly white look

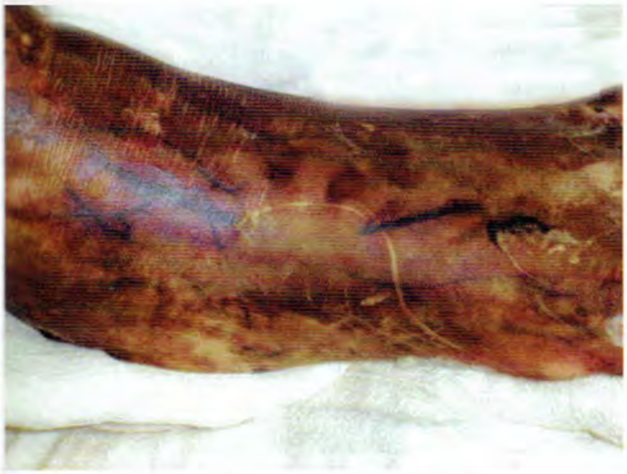


Fig. 14.6B: Deep burns with charred look

inability to pull out hairs indicates otherwise (Fig. 14.7A and B). Deeper a burn injury, greater is the loss of hair follicles, sebaceous and sweat glands. It is the epithelial lining of these epidermal adnexal elements which re-epithelializes the surface. In very deep dermal burns, their number may be very small and any superimposed bacterial infection can convert a partial thickness wound into a full thickness wound hampering the process of spontaneous healing. Thus, it is imperative to prevent this conversion by appropriate wound care. The primary objective in treating a partial thickness burn injury is to provide an environment which inhibits microbial growth and at the same time promotes wound healing. On the other hand, the aim while

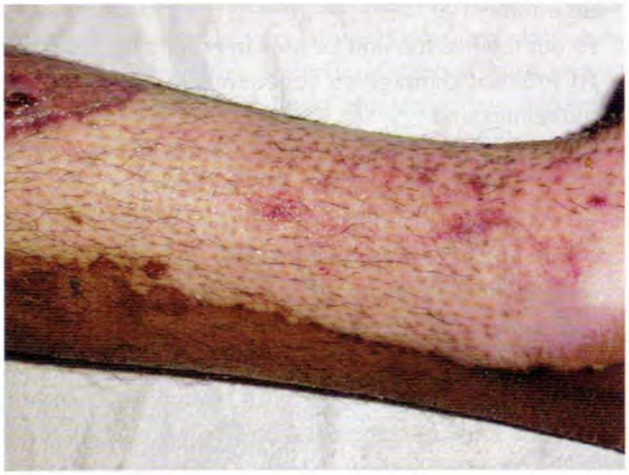


Fig. 14.7A: Superficial partial thickness burns with almost all hair intact–Heals very quickly

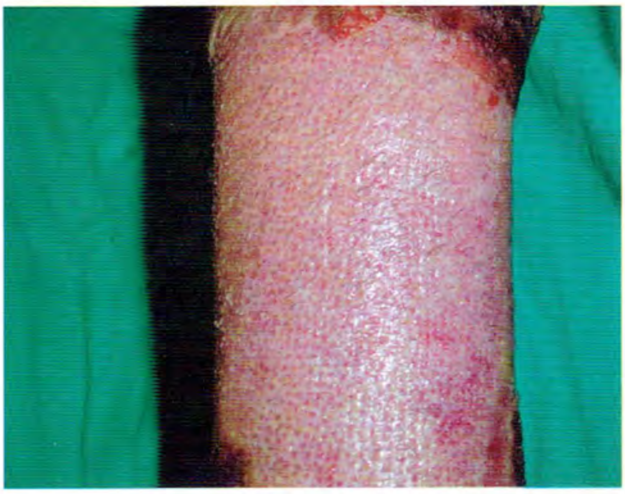


Fig. 14.7B: Superficial partial thickness burn but deeper than Figure 14.7A, with loss of some hairs. Remaining hair allowing epithelialization–Epithelial buds seen

treating a full thickness burn is early removal of dead tissue, preventing/minimizing and localizing infection to the burn wound and closure of raw area at the earliest with skin grafts.

Aims of Local Burn Wound Care

The basic aims of local burn wound care are:

1. To clean debris and particles from the burn wound
2. To prevent damage to remaining viable epithelium
3. To produce an environment which inhibits microbial growth
4. To encourage slough separation by natural means
5. To allow free drainage of exudates from the wound and prevent them from coming in contact with the environment
6. *To immobilize the injured area in order to reduce pain*
7. To prevent damage to regenerating, delicate neo-epithelium, and
8. To prevent development of bacteremia and septicemia.

Since minor burns (<10% in children and <15 % in adults) do not require intravenous fluid replacement, the local wound care can be instituted as soon as the patient arrives. However, in moderate to severe burns (> 10% TBSA in children and > 15-20% TBSA in adults) where intravenous fluid replacement and other emergency steps for resuscitation are essential, the burn wound care has to be delayed for sometime and till then, the wounds should be covered with a sterile sheet to reduce the chances of wound contamination.

Principles of Wound Management

The **principles of wound management** which should be kept in mind while treating burn wounds are as follows[1]:

Debridement

Debridement involving removal of dead material within a wound to improve normal healing is a very essential step for burn wounds. Thermal injury involves necrosis of skin and subcutaneous tissues to varying depths which results in formation of eschar which releases chemical mediators which damage local capillaries and epithelial elements though they also stimulate leucocytes to digest debris. As a result, if this necrotic tissue is not removed early, it prolongs inflammation and retards epithelialization and hence healing of wounds (Fig. 14.8).

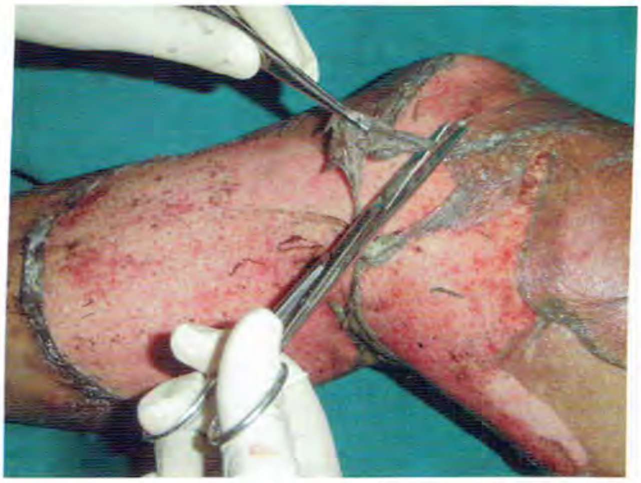


Fig. 14.8: Debridement of dead skin

During World War I, the battlefield surgeons realized that regular excision of dead tissue from war wounds reduced the incidence of infection and accelerated healing process.[2] This concept is now used as a fundamental principle while managing burn wounds worldwide. Debridement of burn wound is different from excisional surgery. It involves washing and gradual removal of loose debris and necrotic tissue and handling of blisters, which is usually done bedside or in the dressing room without anesthesia. Excisional surgery, on the other hand, is excision of the burn wound (surgical removal of the eschar) carried out in the operation theater in order to prepare the wound for early skin grafting.

Oxygenation of Tissues

A continuous supply of oxygen is essential for all phases of wound healing. Following burns, the blood supply to the burn wound is reduced because of edema and impaired circulation leading to poor oxygenation of tissues. This reduced tissue oxygenation not only impairs wound healing but also favors bacterial invasion of the wound. The result is that the wound heals poorly and with excessive scarring. To counter this, some burn centers have used hyperbaric oxygen to assist wound healing. However, it has not been used widely.[3,4]

Simpler measures can also be taken to ensure adequate oxygenation of burn wounds (a) the burned extremities should be elevated, (b) constricting dressings and casts should be avoided, (c) early escharotomies/fasciotomies should be done to avoid compartment syndrome in case of circumferential full thickness burns (Figs 14.9A to C), (d) early ambulation with full range of joint motions to be started and e) removal of dead tissue.

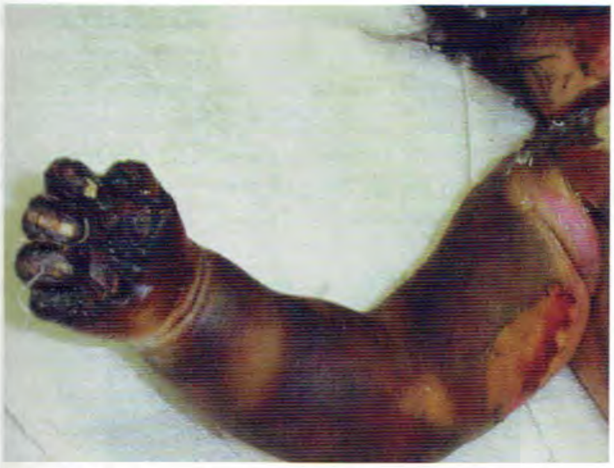


Fig. 14.9A: Circumferential full thickness burns in an infant, compromising circulation to the hand

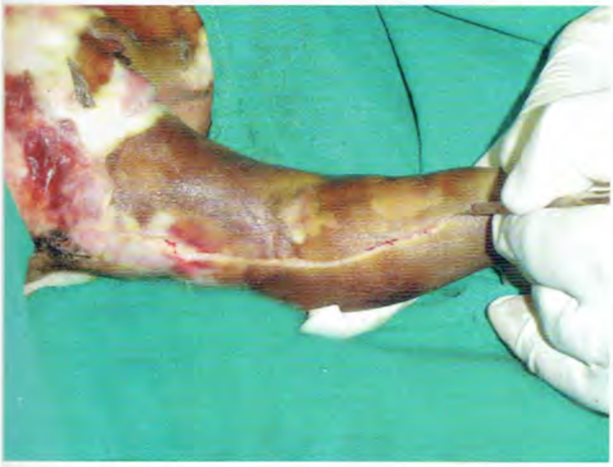


Fig. 14.9B: Incision through the eschar—Escharotomy

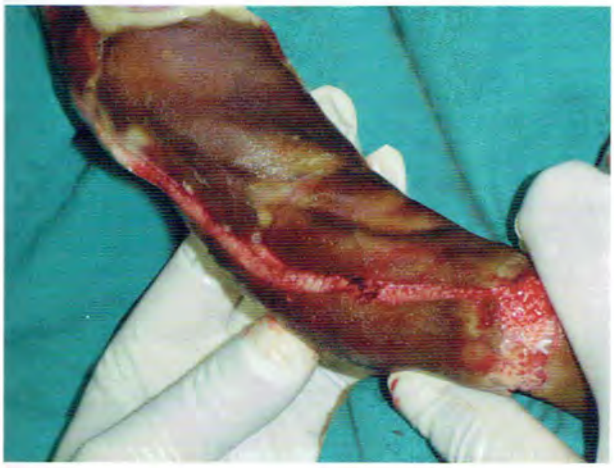


Fig. 14.9C: Tissues underneath the eschar bleed once escharotomy is done

Epithelialization

In all partial thickness burns (other than first degree epidermal burns), the epidermis is completely destroyed. However, the epidermal appendages, i.e. the hair follicles, sweat glands and sebaceous glands are spared. It is from these surviving epithelial structures that the epithelium spreads rapidly to provide a new intact epithelial surface which is elastic and supple. In case of deep dermal burns the majority of the dermis and all sebaceous glands are destroyed and only deeper parts of hair follicles or sweat glands survive. As the eschar separates, epithelial buds emerge from the site of these remaining epidermal appendages and from these tiny islands the new epidermis proliferates over the wound surface (Fig. 14.10). As this re-epithelialization is very slow, the healed skin shows hypertrophic scarring. Superficial dermal burn wounds should be managed with moist protective dressings which enhance epithelial growth. Exposure technique which involves exposure of burns to air leads to formation of a scab which retards epithelial migration and hence healing. In recent years, a number of synthetic, occlusive and semi-occlusive dressings have been developed and used for superficial burns and donor areas.[5,6] These have been found to protect the fragile new epithelium during early phase of healing and thus hasten wound healing. Burn wounds should be closely watched for development of infection because the latter can increase the depth of wounds.

Formation of Granulation Tissue

In case of very deep dermal and full thickness burns, in the absence of infection, the dead tissues become dry, firm and

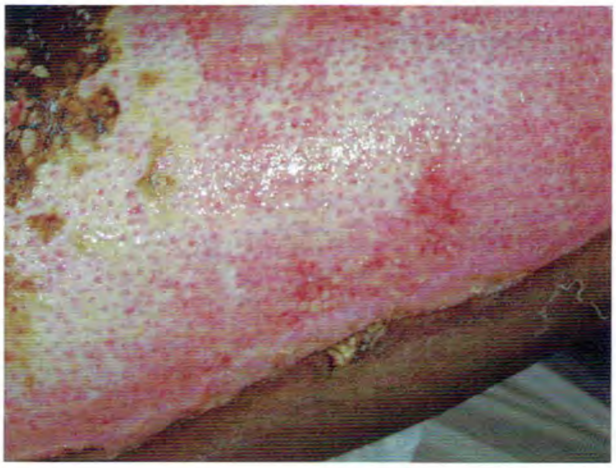


Fig. 14.10: Epithelialization occurring underneath the eschar–Seen once the slough is removed

black called eschar. Beneath this eschar, cellular and capillary activity in the underlying viable tissues produces a layer of red granulation tissue which is exposed when the eschar separates. The granulation tissue is totally devoid of epithelial cells so that the healing of a burn raw area can take place only by epithelial migration from the normal surrounding epithelial edge and wound contraction. Initially this ingrowth is rapid but only for a certain distance after which the cells stop growing. Over flexor aspects of joints or in areas where the skin is loose and contraction is possible, the wound closes of its own with development of contractures. In areas where the underlying bone(s) prevent contraction, the granulation tissue remains as a non-healing wound till it is covered with a skin graft/flap (Fig. 14.11).

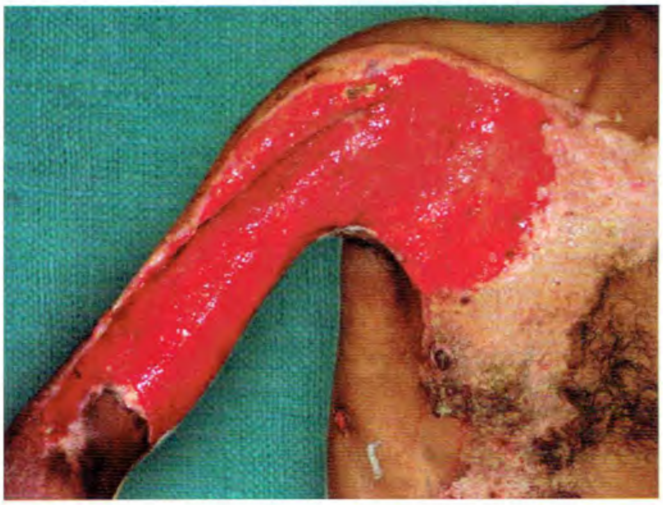


Fig. 14.11: Granulating wound, shows that epithelialization from the margins stops after a certain distance

Wound Contraction

In case of untreated/conservatively treated deep burn wounds, the granulation tissue forms on the raw areas and epithelialization begins from the margin of the raw areas. At the same time, there is laying down of collagen fibers under the granulation tissue. These areas, if not grafted at the earliest, go on to develop chronic granulation tissue. Their vascularity reduces, the superficial layers of granulation tissue ooze serum which clots and this is again invaded by inflammatory cells and capillaries to form fresh granulation tissue. As time passes, the deeper layers progressively mature into fibrous scar tissue with overlying thin granulations. This begins to contract in an effort to close the raw areas. It is this contraction of the bed which leads to severe contractures causing severe deformities in which will then require correction by release and gr (Figs 14.12A and B) which will then require corre release and grafting. To avoid this, a burn surgeon c them with skin grafts as soon as the patient is fit for su and the area is fit for an optimal graft 'take', thus li the formation of scar.

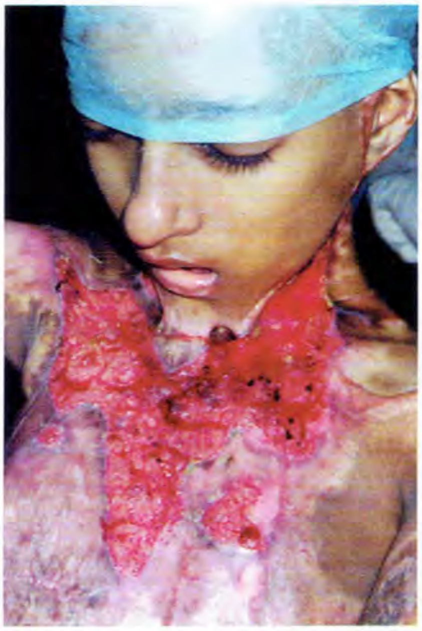


Fig. 14.12A: Wound contraction —If surgical intervention is no the wound bed contracts leading to contracture of neck

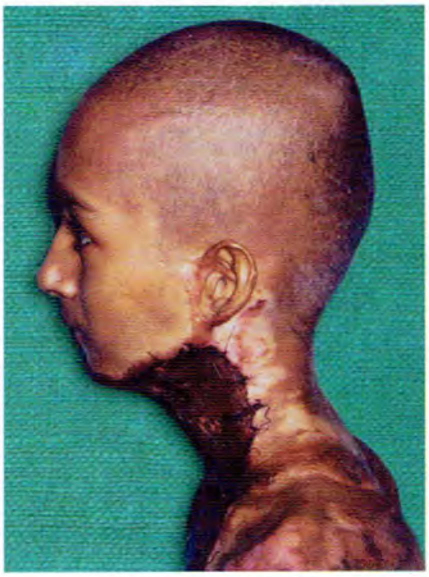


Fig. 14.12B: In same patient —Surgically released neck with split skin grafting

INITIAL CARE OF BURN WOUND

Once the resuscitation procedures have been initiated, the burnt area has to be evaluated in detail for its extent and depth. It is preferable to shift the patient to the specified dressing room where the wound can be managed with all aseptic precautions. This is important because immediately following burns the skin surface is sterile as all surface bacteria are killed by the heat. One should avoid introducing exogenous bacteria to the wound by all means in order to avoid burn wound infection. All the loose nonviable tissues should be removed with minimum trauma. The area should be thoroughly washed with Savlon® and then normal saline to remove all the debris. Only after thorough debridement, a proper appraisal of depth of burns can be done and it should be properly charted in the "Burn Chart" for future reference (Fig. 14.13). This chart contains detailed observations of the wound along with any surgical procedure performed. Any change in burn wound depth can also be charted in the same form at a later date to follow the progress of the burn wound. All encircling bracelets, rings and other jewellery on the person of the burn victim should be removed because they can act as tourniquet once edema sets in, causing distal ischemia.

Management of Blisters

Management of blisters is very controversial. Theoretically, the fluid in an intact blister is sterile. However, it is rich in proteins which cause it to set into a gelatinous form which is an ideal culture medium for bacterial growth if the fluid happens to get contaminated. This fluid retards the local immunity by impairing the normal function of inflammatory cells and also retards the fibrinolytic process thus, delaying the wound healing.[7] The controversy about blisters is (a) whether they should be ruptured and deroofed or else kept intact, and (b) whether the blister skin acts as a skin substitute or not. While very small blisters(< 5 cm) may be allowed to be kept intact, the larger blisters (> 5 cm)run the risk of rupture at the time of initial dressing or else may get ruptured in odd situations and the burn wound gets contaminated. It is very difficult to keep blister fluid sterile for a long time and so it has been advocated to at least aspirate them with a needle to allow the blister membrane to stick to the wound. Other workers consider the blister skin as dead and necrotic tissue and hence deroof the blister and dress the wound (Figs 14.14A and B). In children, it is preferable to rupture the blisters because they are bound to get ruptured soon due to their uncontrolled activity.

After cleansing, the wounds are dabbed dry with sterile gauze and then the appropriate method of further wound management is selected, i.e. exposure method (open method) or closed method (occlusive method) of dressing. In most patients, neither of these methods used alone is ideal for local care and frequently both methods are used in different areas of the body in the same patient for best results.

Exposure or Open Technique

The open method of burn treatment has been used since eternity but was reintroduced by Wallace in 1948.[8] It is especially useful for burns of face and perineum which cannot be adequately dressed. Extensive non-salvageable burns more than 90 % TBSA may also be managed by this method decreasing the requirement for manpower, material and cost. This technique consists of placing the patient on a clean, dry, sterile sheet after cleaning and debriding the wounds. The wounds are left exposed to air without any topical agents or any covering agents. The exudate from the wounds dries up and along with the layers of destroyed skin forms a scab or an eschar. The latter acts as a 'dressing' protecting the underlying tissues from bacterial colonization (Fig. 14.15A). Although the dry cool air inhibits bacterial growth, pathogenic organisms can grow from the deep surface of the crust. Under these circumstances, the activity of the organisms is usually low and the body is able to fight them and hence, healing can take place. The drying process, however, may retard the spread of epithelium which is faster under moist conditions.

For deeper wounds, open treatment may be less satisfactory as the dry eschar takes a long time to separate. The patient and his attendants also may find the exposed wounds unsightly and uncomfortable. This method is not very suitable for wounds over joints as the scabs over a joint surface may crack repeatedly and bleed. In case of hands, exposure method allows full range of movements at all joints which avoids deformities of the hand.

A modification of the open technique is to apply a thin layer of antibiotic ointment over the wounds after cleansing and drying them daily, especially on the face. Sometimes an antimicrobial impregnated gauze is used on exposed wounds and this gauze acts as a wick to transport the exudates to the gauze surface where it dries up forming a scab. The advantage with this technique is that the scab does not adhere to the wound and the gauze can be removed easily

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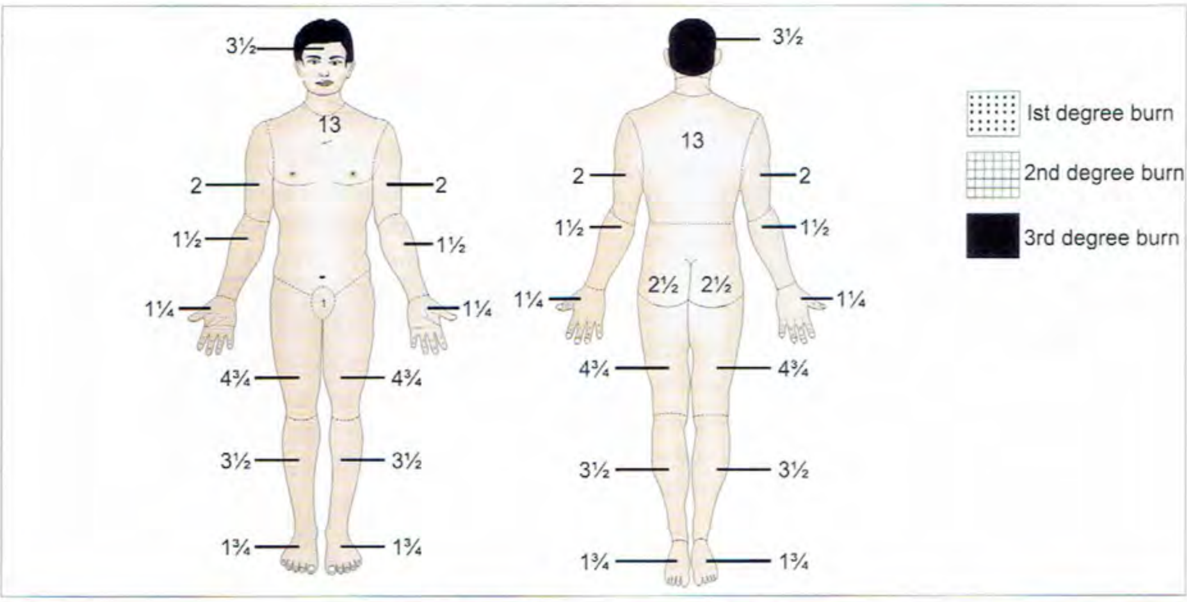
Name	____________	M.R.D.No.	____________
Age	____________	Index No. B	____________
Weight	____________	Sex	____________
		Date of observation	____________

Estimating the area burned



Area	Birth 1-yr	1-4 yr	5-9 yr	10-14 yr	15 yr	Abult	Partial-thickness second degree	Full-thinckness third degree	Total
Head	19	17	13	11	9	7			
Neck	2	2	2	2	2	2			
Anterior trunk++	13	13	13	13	13	13			
Posterior trunk+	13	13	13	13	13	13			
Right buttock	2½	2½	2½	2½	2½	2½			
Left buttock	2½	2½	2½	2½	2½	2½			
Genitalia	1	1	1	1	1	1			
Right upper arm	4	4	4	4	4	4			
Left upper arm	4	4	4	4	4	4			
Right lower arm	3	3	3	3	3	3			
Left lower arm	3	3	3	3	3	3			
Right hand	2½	2½	2½	2½	2½	2½			
Left hand	2½	2½	2½	2½	2½	2½			
Right thigh	5½	6½	8	8½	9	9½			
Left thigh	5½	6½	8	8½	9	9½			
Right leg	5	5	5½	6	6½	7			
Left leg	5	5	5½	6	6½	7			
Right foot	3½	3½	3½	3½	3½	3½			
Left foot	3½	3½	3½	3½	3½	3½			
Total									

Fig. 14.13: Burn chart used in the author's unit–Depth of burns in different areas can be charted

without the risk of bleeding from the wound or else damage to the newly formed epithelium (Fig. 14.15B).

If the burned area is superficial partial thickness, the whole scab/gauze begins to separate after a week or so and this is trimmed gently and repeatedly with scissors and finally comes out in 15-21 days to leave a well healed skin surface. If the scab does not separate by 3 weeks, the burn is treated as a full thickness burn and the management is changed to the technique of occlusive dressings which hasten separation of eschar so that the raw area can be split skin grafted. For patients treated by exposure method, appropriate maintenance of the environmental temperature and humidity is very important. This is because leaving the wounds exposed increases the evaporative losses as well as substantial body heat loss. To compensate for this, the ambient temperature must be maintained in the range of 28-32 °C and humidity at about 40 %.

The advantages of open method are:

- Wound examination is easier
- Earlier detection of wound infection
- Early physiotherapy
- Easier control of hyperpyrexia, and
- Wound environment is not conducive for bacterial colonization.

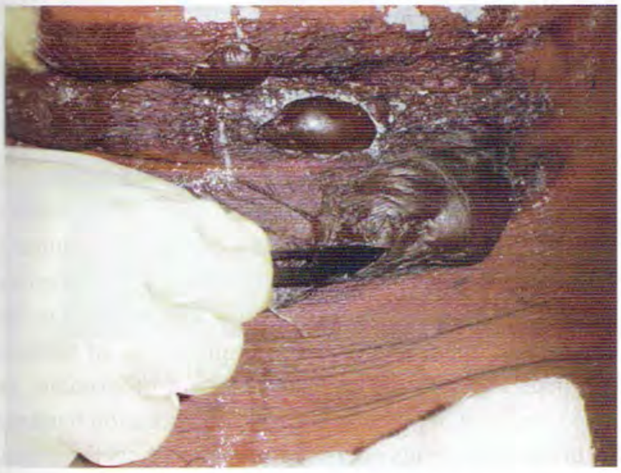


Fig. 14.14A: Blister being incised

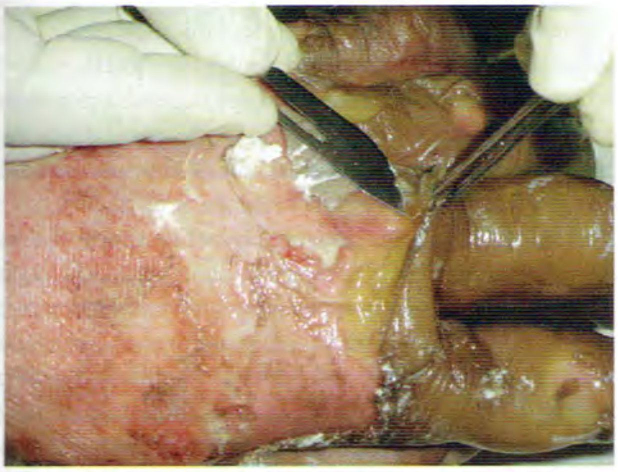


Fig. 14.14B: Deroofing of blister done surgically—Underlying coagulum seen

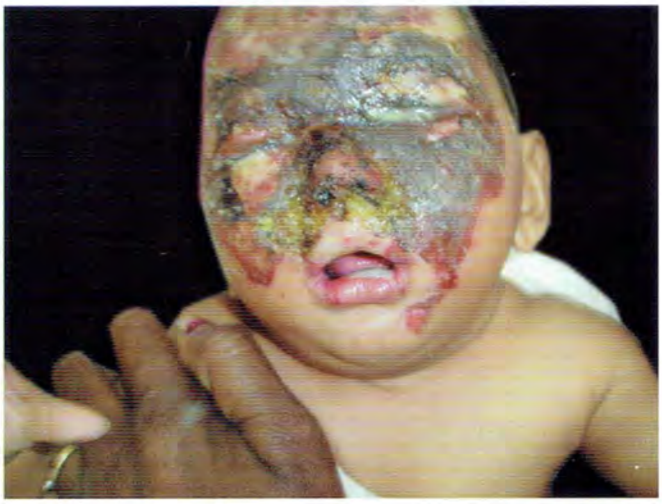


Fig. 14.15A: Facial wounds treated by exposure technique –Exudates from the wound dries up and forms a scab which acts as an external dressing to protect underlying tissues from infection

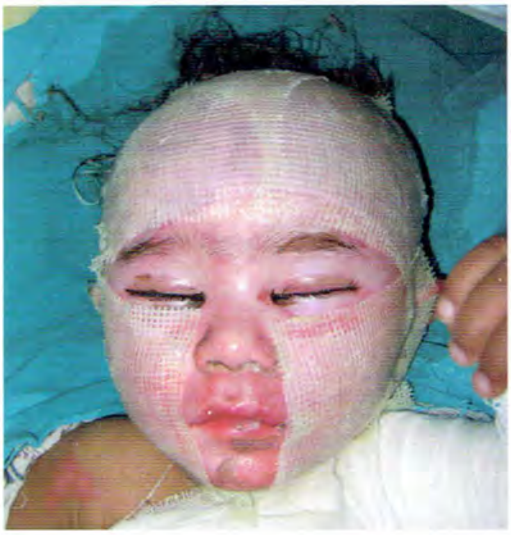


Fig. 14.15B: Exposure technique but with application of antibiotic impregnated paraffin gauze—Transports exudates to the surface of the gauze so that the scab does not adhere to wound surface and gauze can be removed easily without causing pain or bleeding from wound surface

The disadvantages of open method are:

- Delay in eschar separation
- Evaporative losses increase
- Chances of hypothermia are more because of loss of body heat, and
- Not possible where isolation is not available.

Occlusive Dressing or Closed Technique

This method is most useful for patients who are treated on outpatient basis, patients who need to be transported from one center to another, circumferential burns of extremities and torso, in infants and children and uncooperative adults (Fig. 14.16). Occlusive dressings are avoided in burns of face and neck and perineal burns.

The advantages of occlusive dressings are:

- It reduces evaporative losses
- Provides a mechanical barrier to prevent bacteria from contaminating the wounds
- Absorbs exudates from the wound surface
- Assures maintenance of functional position in burned extremities
- Maintains moist environment which encourages epithelialization
- Patient is more comfortable
- Provides splintage to joints thus preventing contractures.

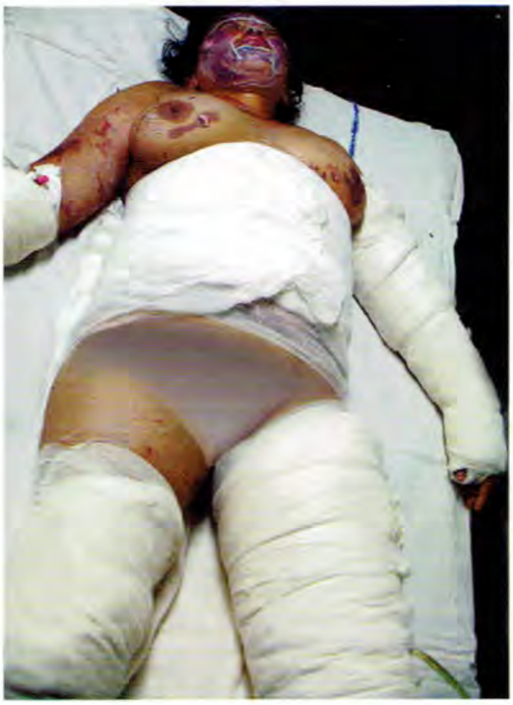


Fig. 14.16: Closed dressing technique

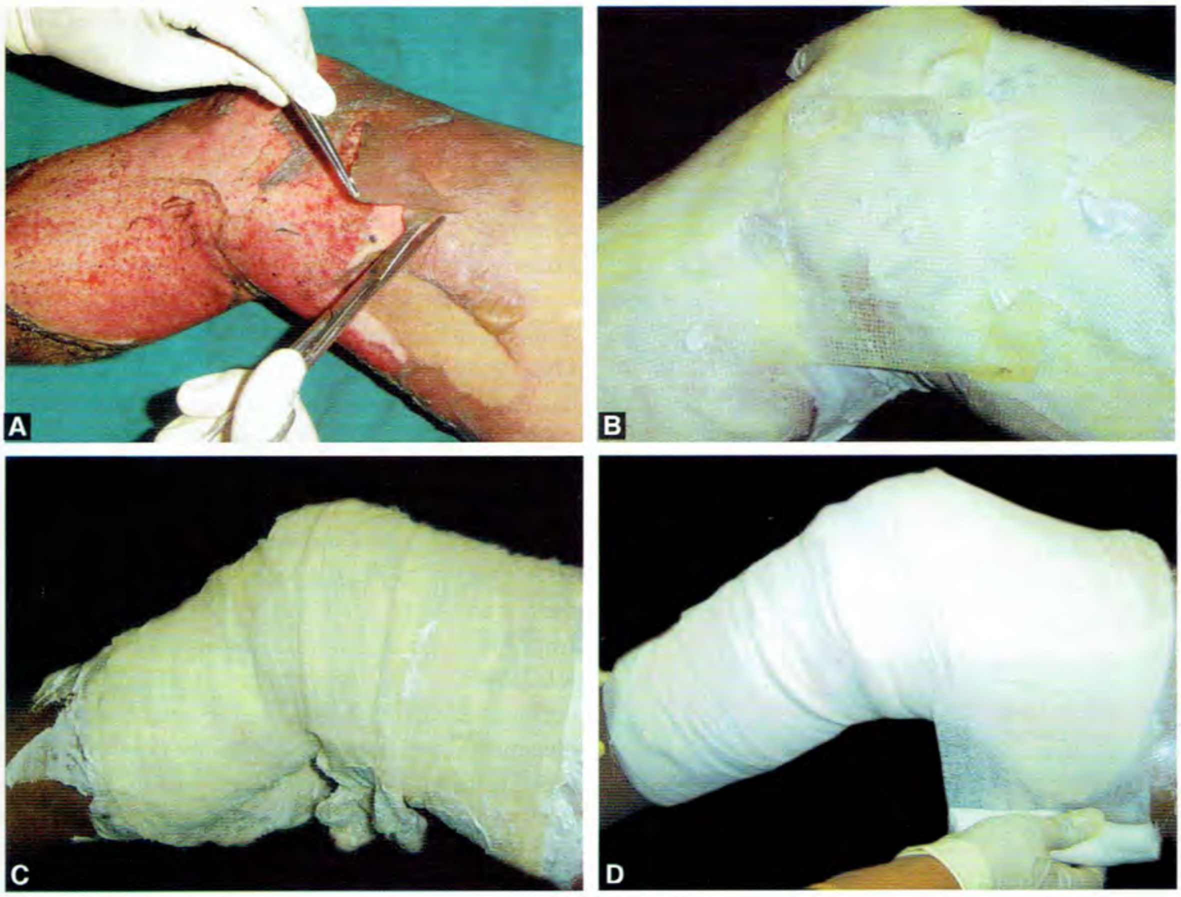
Dressing Technique

The technique of dressing a burn wound is very importan for the final outcome of burn wound treatment program All burn dressings should be done with proper aseptic precautions to prevent wound contamination. All doctor and nursing staff should be properly gowned and gloved Majority of the dressings can be performed without genera anesthesia. Usually sedation with opioid analgesics (e.g morphine) is adequate.

A satisfactory burn dressing is one which can absorb al the fluid exuded from the burn wound and distributed evenly throughout so that there is no collection of the exudate on the wound surface. The material which fulfills these requirements is the Gamgee pads which consists of cotton wool sandwiched between two layers of absorbent gauze These are easy to prepare and can be made in assorted sizes suitable for small as well as extensive areas. These are prepared, sterilized and kept ready to use so that they are available at all times.

The innermost layer of occlusive dressing consists of a layer of an antimicrobial agent over which a wide mesh gauze impregnated with paraffin or petroleum jelly is applied This tulle grass is non-adherent so that pain and trauma to the regenerating neoepithelium can be minimized at the time of change of dressing. After the non-adherent gauze and antimicrobial agent is applied, multiple layers of Gamgee pads, depending on the expected amount of exudate, are applied and kept in place with gentle compression bandage This dressing prevents edema formation, splints the wound and facilitates absorption of discharge. However, it should not be constricting in any way. This outer layer of the dressing should be porous to allow evaporation of water and prevent inner layers from becoming soggy as tha encourages microbial growth (Figs 14.17A to D).

The frequency of dressing change depends upon depth of burns and degree of infection in burn wound. In case o partial thickness burns when there are no signs of infection the first dressing can be left intact for 5 days. In case o full thickness burns, the dressing should be changed every 3 days. Earlier change of dressing in either case is indicated if there is soakage in dressing or any sign of infection o burn wound which includes local pain, unexplained fever foul smell from dressing and increased purulent discharge from wound. In these cases frequency of dressing change can vary from daily dressing to alternate day dressing til infection is controlled. The topical agent being used on wound should also be changed if discharge increases. There



Figs 14.17A to D: (A) Removal of loose dead skin. (B) Application of silver sulfadiazine over paraffin gauze. (C) Application of gauze and Gamgee. (D) Light compressive bandage of the limb

are various antimicrobial agents used for burn dressings which are discussed later in this chapter.

Biological Dressings

The use of topical antimicrobial creams for burn wound dressing has been in vogue for about half a century. Their use for treatment of partial thickness burns was favored by all surgeons as they allowed better control of burn wound sepsis. However, the application of topical creams requires frequent dressing change which causes pain and anxiety to the patient. These drawbacks instigated the research for alternative treatment with temporary skin substitutes or biological dressing materials. The application of biological dressings is a good alternative to daily change of conventional dressing with antimicrobial agents for superficial burns. Though their use improves the quality of care given to burn patients, they are expensive and their use should be rationalized. Prospective studies have shown beneficial effects of biological dressings in superficial burns even though the final outcome is the same. Pain is absent or reduced significantly, patient is more comfortable and discharged from the hospital sooner. Hence, the overall morbidity is reduced. The most commonly used temporary skin substitutes are collagen dressings, Biobrane®, Integra® etc. Their use and application are described in detail in the chapter on Biological dressings.

Escharotomy/Fasciotomy

In full thickness circumferential burns of limbs, neck and chest, the tight dry eschar with underlying edema may produce a tourniquet effect. This has to be diagnosed early in order to avoid vascular compromise to distal extremities and respiratory impairment. To diagnose distal vascular compromise, the following clinical signs and symptoms are looked for: cyanosis of distal unburned skin, delayed capillary filling in nails of fingers or toes distal to circumferential burns in limbs, paresthesias or deep tissue pain. All these signs warrant a second look at the burn wound and diagnosis can be confirmed by a color Doppler to look for arterial flow in distal palmar arch for upper limb and posterior tibial artery in lower limb. These circumferential burns are treated by performing an escharotomy (Fig. 14.18) wherein an incision is made through the full thickness of the eschar releasing the constricting effect and thus, decompressing the underlying structures and improving the distal blood supply. In case escharotomy does not give adequate decompression, as in case of high voltage electrical injury where the muscles are also involved leading to edema under the deep fascia and raising the compartment pressure, the incision has to be extended till the underlying deep fascia (fasciotomy) in order to release the compartment pressure (Fig. 14.18B).

Technique: The procedure is done bedside using sterile technique (Fig. 14.18B). Anesthesia is not required as the eschar is dead tissue and hence painless. Sites for incisio for escharotomy are given in Figures 14.19A and B, taki care to avoid injury to major peripheral nerves. The incisi has to be first given in the midlateral line across involv joints and throughout the length and thickness of the esch If perfusion does not improve, midmedial incision is giv to increase the decompression. Blood loss is usually minim because incision goes through the avascular eschar only

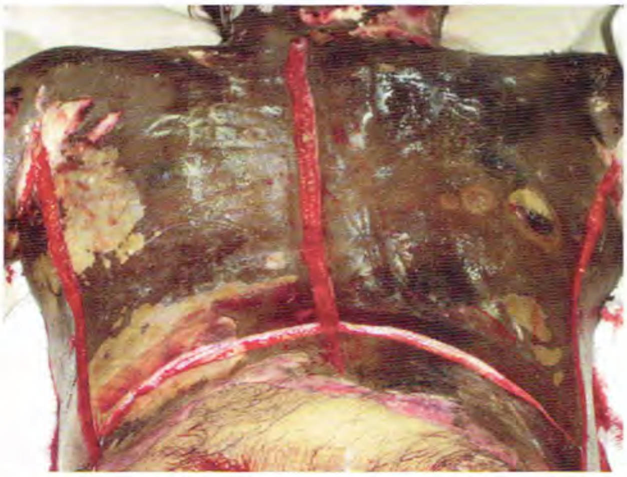


Fig. 14.18A: Escharotomy incisions for full thickness circumferential burns of chest restricting chest expansion

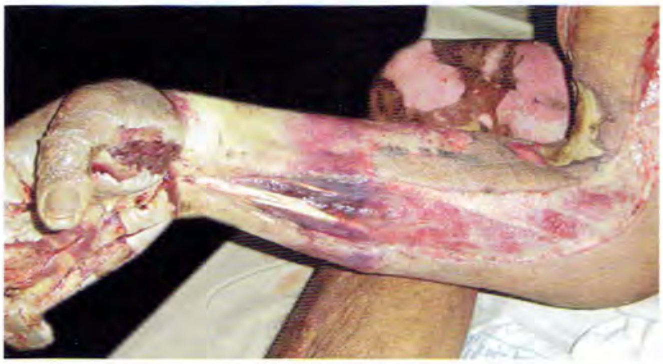
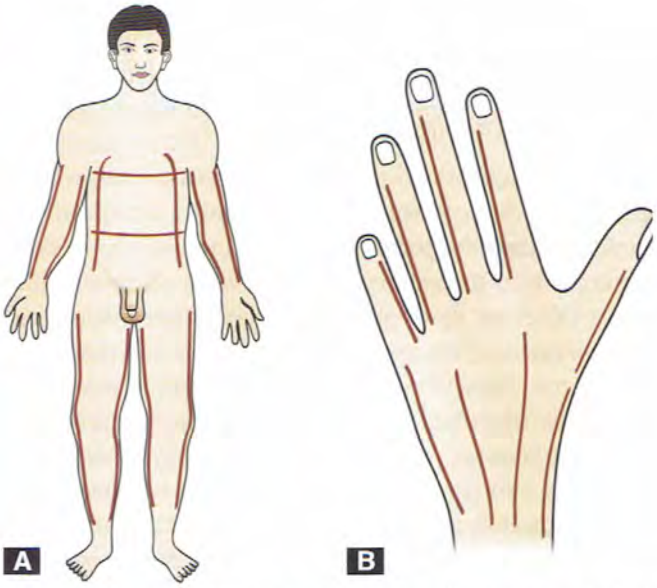


Fig. 14.18B: Fasciotomy incision over arm in electrical contact burn

In case of electrical burns when there is edema of t muscles which increases the compartment pressure fasciotomy is required (Figs 14.20A to D). In these case the skin of the limb may be unburnt and requires loc anesthesia before giving the fasciotomy incisions in differe compartments. At times, the carpal tunnel has to be open in the hand to release the pressure on the nerves (Figs 14.21 and B). In that case, the incision can be extended over t forearm as an S-shaped incision over the flexor aspect the forearm and hand. Fasciotomy incisions should be give from proximal to distal direction so that the pressure released gradually and immediate profuse bleeding does n occur interfering with the incisions. If there is significa bleeding during fasciotomy, it can be controlled by pressur dressing for a brief duration or by using electrocautery.

Since the incisions expose the underlying healthy tissue to a risk of infection by bacteria colonizing the burn woun it is important to give cover by topical antimicrobial age during dressing.



Figs 14.19A and B: (A) Line diagram showing escharotomy incision lines. (B) Line diagram showing escharotomy incisions for hand

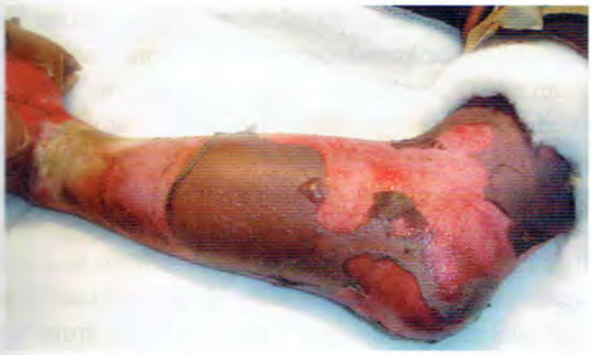


Fig. 14.20A: Electrical contact burn in arm causing gross edema of limb and distal circulation compromised due to compartment syndrome, demanding urgent fasciotomy

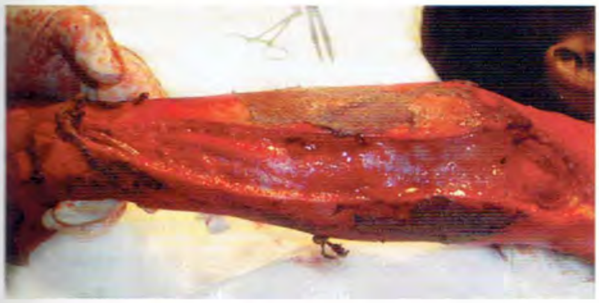


Fig. 14.20B: Incisions given till deep fascia, releasing the compartment pressure

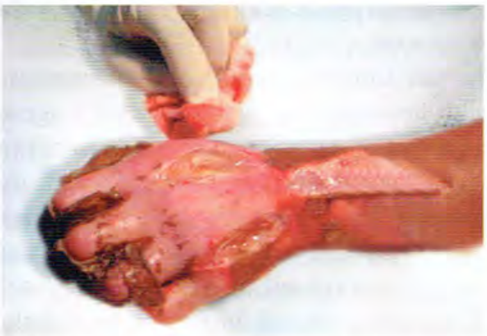


Fig. 14.20C: Fasciotomy incisions over dorsum of hand

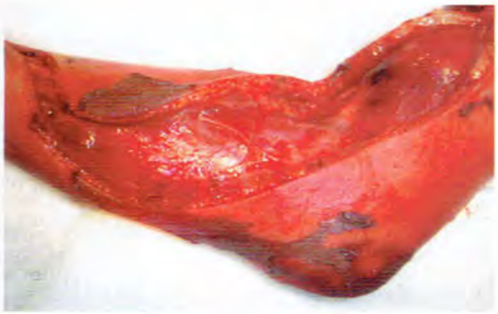


Fig. 14.20D: Following fasciotomy, the underlying muscles bulge out and bleeding on surface is seen

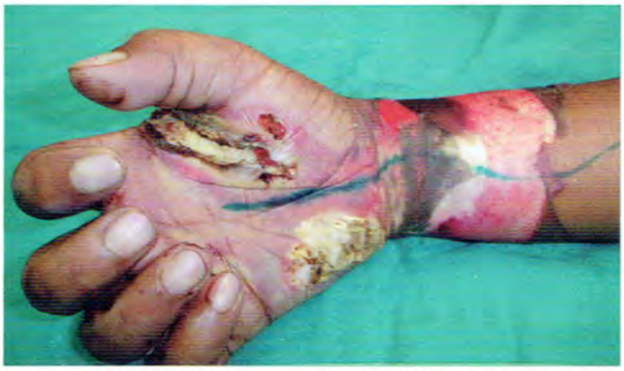


Fig. 14.21A: Incision for opening carpal tunnel to decompress nerves

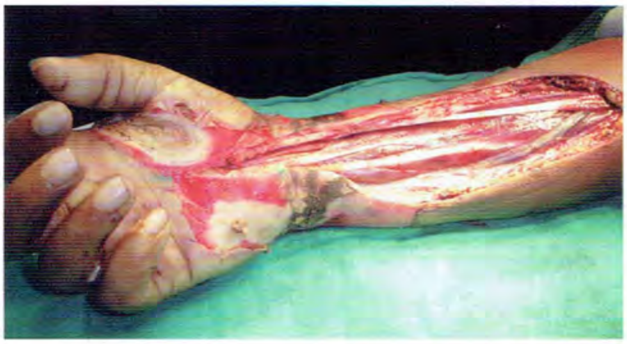


Fig. 14.21B: Incision extended into forearm—Circulation to hand restored and hand saved by timely intervention

Hydrotherapy

Hydrotherapy involves washing the burn wound or giving a bath to the entire patient prior to dressing change. This can be given in the form of a shower or immersion in saline bath (Arzo bath tub) or Hubbard tank (Figs 14.22A and B). The advantages of hydrotherapy are:

- It improves general hygiene and decreases the number of bacteria
- Patient has a feeling of well-being
- Dressings can be removed easily and wounds cleaned painlessly
- Active exercises can be performed during hydrotherapy which prevents stiffness of joints

Ambulatory patients are managed by using shower bath whereas non-ambulatory patients or those with extensive burns are best managed in a saline bath or Hubbard tank. Minor debridements can also be carried out during this procedure. The main disadvantage of using bath tubs or tanks is the risk of cross contamination amongst the patients. Additionally, this is a cumbersome procedure involving a team of doctors, nurses and therapists.

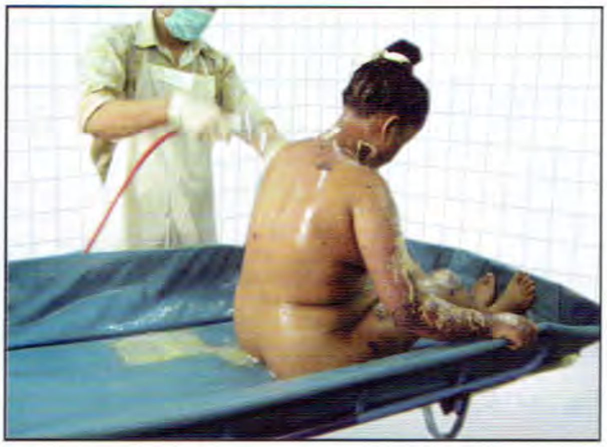


Fig. 14.22A: Shower bath on a shower trolley

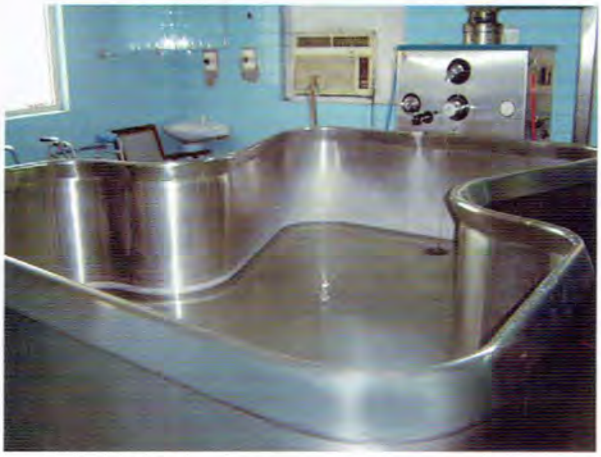


Fig. 14.22B: Saline bath tub used in the author's unit

TOPICAL ANTIMICROBIAL THERAPY IN THE TREATMENT OF BURN WOUND

A variety of local medicaments have been used on burn wounds since eternity,which ranged from tinctures (600 BC), vinegar soaked dressing (430 BC), greasy dressing (1596), tannic acid by Edward Clark Davidson (1894-1933) to Gentian violet by Aldridge in 1933 to Petroleum gauze by Harvey Allen in 1942. All these methods, however, fell into disrepute because of local and systemic toxicity or ineffectiveness of these substances. This initiated a research into finding newer topical agents which are more effective and have fewer side effects.

History of Topical Antimicrobial Agents

The topical therapy of burn wounds dates back to the beginnings of civilization. Throughout history, a number of famous philosophers, physicians and scientists ha contributed to the knowledge of burn wound manageme including such notables as Hippocrates, Celsus, Pliny t Elder, Galen, Aristotle, Rhases, Clowes, Paré, Hildan Marjolin, Dupuytren and Syme. A diverse variety substances have been advocated as effective topical bu treatments including various plants, gums, milk (goat mi and milk from "a women who has given birth to a son tea leaves, roasted angle worms, oak bark extract, hon cork, bear fat, bran, ashes, vinegar, wine, fat from "ve old wild hogs," calcium chloride soaks, moss "from t skull of a person hung," red sandalwood, cold water, sali baths, lemon strips soaked in oily dressings, soot, spid webs, linseed oil mixed with lime water, picric aci medicated paraffin, carbolic acid, cod liver oil and "portio of a genuine mummy." Over the years, the goals of topic burn treatment have changed. By the beginning of the 2 century, it was believed that the systemic derangeme seen in burn patients resulted from a release of 'toxi from the burn wound and thus, the goal of topical thera would be to bind or leach toxins before they can absorbed.[11] A secondary goal was to dry out the burn wou or to create a hard coagulum to minimize exudate, draina or fluid loss. A number of "escharotics", including 5% 10% silver nitrate solution, gentian violet,[12, 13] "triple" dy trinitrophenol and tannic acid were employed, either alo or in combination.

There was a drastic change in the treatment of bu wound with dressings along with an antiseptic agent in 18 following discovery of the concept of antiseptics by List Carbolic acid, mercurial compounds and aniline dyes we used as inner layer of burn dressing. Unfortunately, th were unselective in their action and damaged normal tissu along with bacteria colonizing the wound surface. Henc their use was discontinued. In the mid-twentieth centu phenol containing dressings were highly recommended.

Davidson at the Henry Ford Hospital first described t use of tannic acid spray in 1925,[12,14] and believed that th modality reduced pain and produced a cleaner wound bed. In 1935, Bettman added 10% silver nitrate to a 2.5% tann acid solution, which allowed wound tanning to occur seconds.[11] Reports of liver necrosis in patients treated wi tannic acid began to appear in the 1930s, although the liv necrosis was often ascribed to the burn injury itsel Additionally, pus tended to accumulate under the coagulu formed by tannic acid application. McClure (1944) [12], al from Henry Ford Hospital, reported that tannic acid was hepatotoxin and the use of this agent was discontinued.

Work by Aldrich, Teplitz, Lindberg and others established that septicemia in burn patients, a major cause of mortality, frequently originated in the burn wound.[13] Attention now turned to the application of various topical antimicrobials to the burn surface and the development of mafenide (Sulfamylon®), silver nitrate, silver sulfadiazine and others revolutionized burn care.

In early part of World War II, Marfanil which was later called Mafenide was used by German army and was found to reduce the incidence of gas gangrene. Subsequently, it was used by US Army for traumatic wounds as it was found to be effective against clostridial and enteric organisms. Its efficacy in controlling burn wound sepsis was discovered by Moncrief in 1964. Although it was found to be very effective against *Pseudomonas* especially in treatment of established infections, it was relatively inactive against gram-positive organisms, especially streptococci which was the main cause of burn wound infection at that time.

In 1965, Moyer and colleagues showed excellent prophylactic use of 0.5% Silver nitrate against *Psuedomonas*. But it had no role in established infections and also caused dys-electrolytemia and the patient was uncomfortable because of the black staining and continuously wet feeling. So its use fell into disrepute.

In 1968, a new antimicrobial, namely silver Sulfadiazine, was discovered by Fox for use in burns. Even after half a century later, no serious side effects have been reported despite its widespread use.

Many other antibiotics and antiseptics have been used singly and in various combinations for burn dressing, namely, gentamicin, neomycin, bacitracin, polymyxin, framycetin, nitrofurazone and chlorhexidine, etc. Many of these have proved useful under certain circumstances. However, they have all failed to prevent serious systemic infections in extensive burns, especially *Pseudomonas* infection, which is now the most common organism responsible for burn wound sepsis worldwide.

Basis for Topical Therapy

Immediately following burns, the surface of the burnt skin is sterile and the only organisms present are those present deep in the skin appendages and these are not very pathogenic. Some of these organisms come to the surface following burn but a sterile dressing and use of an antimicrobial agent can kill these organisms. In case of closed dressings, presence of exudates in the dressings, warmth and lack of light under the dressing are ideal conditions for proliferation of organisms after they gain entry to the skin surface through exogenous or endogenous routes, resulting in infection. This can be avoided by using a broad spectrum antimicrobial agent which has action against both gram-positive and gram-negative organisms. The topical agents used for burn wound dressing have a dual purpose –first to control any organisms on the surface of the burn wound and secondly to prevent entry of new organisms from the exterior.

By the end of first week post burn, the deeper surface of eschar especially around the hair follicles shows active proliferation of organisms which can penetrate into deeper tissues and adjacent viable tissue as well, if not controlled. This can again be checked by the application of topical antimicrobial agents which have a good eschar penetration. The deeply burned areas are avascular[10] which not only provide a favorable environment for bacterial proliferation but also prevent penetration of antibiotics administered systemically and thus, ineffective delivery of antimicrobial to the burn wound surface to fight the surface contamination. This is better tackled by topical antimicrobial agents and obviates the need for systemic antibiotics. It is very important to understand that topical therapy does not sterilize the burn wound. It only controls the bacterial proliferation and confines it within the limits of the burn wound. Topical agents are also no substitute for meticulous wound care which includes proper debridement of devitalized tissues which favors bacterial proliferation and prompt de-roofing of sub-escharotic abscesses. On the other hand, proper and judicious use of topical agents can prevent conversion of superficial burns into deeper wounds and also result in spontaneous healing of wounds which appeared to be deep initially and decrease the incidence of systemic sepsis.

Criteria for a Good Antimicrobial Agent

1. It should have a broad spectrum of activity which includes organisms which are most commonly isolated from the burn wound, e.g. *Pseudomonas aeruginosa, Staphylococcus aureus, Klebsiella* and *Enterococci* etc.
2. Ideally the topical agent should penetrate the eschar and yet not be significantly absorbed systemically.
3. There should be minimal emergence of resistance amongst organisms against the agent. If there is high frequency of acquired resistance against the substance being used, the efficacy of the agent decreases and resistant strains produced are also more virulent.

4. It should cause minimum or no discomfort to the patient.
5. It should be economical and easily available.
6. It should have minimal side effects. Though majority of the agents do get absorbed systemically, their use has to be restricted for only certain specific established infections and on the smallest possible wound areas.
7. It should be economical as in many areas of the world, cost is the primary factor which affects selection of the agent.
8. Application should be simple and easy.

Topical Agents

The topical antimicrobial agents, most frequently used for burn wound dressings, are discussed below. As any one agent is not uniformly efficacious in all the cases, it may be necessary to substitute it with a different agent on all or part of the wounds during the course of treatment at different times.

Silver Sulfadiazine (SSD)[10,12,15-17]

It is a white crystalline, highly insoluble compound synthesized by Fox [15] in 1968 by reacting silver nitrate with sodium sulfadiazine. It is the most frequently used topical agent in the form of a water soluble cream base at 1 % concentration, which provides the same amount of elemental silver as 0.5 % silver nitrate solution (30 m Eq /L).[12] At this concentration, the amount of agent at the surface is more than that necessary *in vitro* to inhibit the growth of sensitive organisms.

Mode of Action

The mechanism of action is supposedly due to both silver ion and sulfadiazine moiety which are released at the wound surface by gradual dissociation. Sulfadiazine is a competitive inhibitor of para-aminobenzoic acid (PABA) which is a metabolic precursor. The ionic silver acts primarily on bacterial cell walls where it induces ultrastructural changes and also reacts with the DNA of the bacteria impairing its replication.[12] It is, therefore, primarily bacteriostatic. It is active *in vitro* against a wide range of organisms which include *Staphylococcus aureus*, *Escherichia coli*, *Proteus*, *Enterobacteriaceae* and Herpes virus. Clinical trials have shown that it has reasonable prophylactic effect against gram-negative bacteria. It delays wound colonization by gram-negative bacteria from 10-14 days in extensive burns.

In spite of the dissociation of the compound there is absorption of the silver from the wound. On the other ha Sulfadiazine levels of 1.5-4 mg/100 ml have been repor in the blood. There is also some grayish silver discolorati of the wound because of minimal precipitation of sil chloride or silver sulfide following oxidation of silver exposure to sunlight.

Mode of application: It is applied as 1% cream to burn wound in a thickness of approximately 0.5 -1 cm eit twice a day in case of exposed treatment or once a day closed method. The substance has to be washed off betwe applications in order to debride the wounds properly.

Advantages

It is an odorless cream, easy to apply and causes mini to no pain on application. It does not stain the skin or lin unlike silver nitrate.

It has intermediate penetration capacity into the b eschar between that of readily absorbable Mafenide a minimally absorbable silver nitrate.

Bacterial resistance to silver sulfadiazine, even af prolonged therapy, has not been reported.

Disadvantages

- Crystalluria has been occasionally reported though of much clinical significance.
- Methemoglobinemia is a well known side effect sulfonamide, although seen very rarely.
- Transient leucopenia occurs in many patients treat with SSD and it occurs 2-3 days after starting treatme This resolves spontaneously within a few days desp continued use of the agent.
- A macular rash is seen in <5 % patients and is a cutane sensitivity reaction to SSD.
- A thin proteinaceous gel called' Pseudoeschar' for on the wound surface after topical application of SS This can confuse an inexperienced observer duri assessment of the depth of burn. However, this can removed easily in sheets.
- With prolonged use of topical SSD, there is a risk sulfadiazine resistant gram-negative bacilli, especia Enterobacteriaceae group, developing plasmid mediat resistance.[16]
- Inhibition of epithelial regeneration with t use of 1% SSD has been demonstrated experimentall

Silver Nitrate Solution (0.5%)

Silver nitrate has been used for the treatment of burn wounds for over a century but fell into disrepute because of side effects since it was used in higher concentrations. Five and 10% silver nitrate solution was used as a caustic and escharotic for burn care in the early 20th century, but was largely abandoned by the time of World War II because infections tended to become trapped under the hard eschar produced. It was reintroduced by Moyer in 1965 as 0.5% solution and later popularized by Monafo as a topical agent for burn wound treatment. Though in 95% strength silver nitrate is still used to cauterize hypertrophic granulation tissues, in a 0.5% concentration it has antimicrobial effect without being histotoxic. 0.5% silver nitrate remains the first-line topical agent in many burn centers. For many years, 0.5% silver nitrate has also been used as a topical agent for patients with toxic epidermal necrolysis (TEN).

Mode of Action

Mode of action is apparently due to the free silver ion which is effective against the common microbial flora seen in the burn wound especially gram-negative bacteria. It is mainly bacteriostatic. There is no known resistance seen to this agent and if properly used, it is very effective as a prophylactic agent. There is minimal absorption of silver through the open wounds into systemic circulation because its salts are insoluble. Instead, there is precipitation of its salts, i.e. silver chloride and silver sulfide which cause the characteristic black discoloration of the burn wounds.

Mode of application: The technique of application of silver nitrate solution as described by Monafo and Moyer [11] remains unchanged even today. After cleaning and debriding the loose tissues in the wound, it is covered with a 8-10 layer thickness of gauze dressing which is then soaked with warm 0.5% silver nitrate solution. The dressings have to be wetted every 2 hours so as to ensure that the concentration does not become caustic (> 2-3 %). The advantage of this bulky wet dressing is that it minimizes evaporative loss. The dressings are changed twice daily and their removal achieves significant debridement of the wound also.

Advantages[13]

- Application of silver nitrate is painless.
- No patient hypersensitivity has been reported to it.
- No resistant organisms have been isolated after its use.
- Its use helps in rapid debridement of the eschar because of twice daily change of the bulky gauze dressing and wound becomes ready for grafting 7-10 days earlier than with other methods.
- Its continuous use decreases evaporative water loss which further helps in decreasing metabolic rate and improves nutritional status of the patient.

Disadvantages

- 0.5% silver nitrate has been found to be ineffective in established burn wound infections and over dead epidermis because of its inability to penetrate the eschar.
- It stains all the linen and floor brown or black which are difficult to remove. Thus, its use necessitates use of disposable linens and stain-resistant floor and wall coverings which can increase the cost of treatment.
- There are severe electrolyte derangements noticed with its use. Use of distilled water as a vehicle for application of silver nitrate leads to a markedly hypotonic solution and it causes absorption of large volumes of distilled water into the tissues and eventually into the blood stream. There is leaching of sodium by reverse mechanism which results in hyponatremia which can lead to convulsions, if very severe. This can be prevented by administering large quantities of sodium chloride orally or intravenously. The precipitation of silver as silver chloride and silver sulfide salt results in hypochloremia which can also be corrected by infusing saline.

There is leaching of potassium and calcium also resulting in hypokalemia and hypocalcemia. Hence, careful monitoring of electrolyte levels is required in order to assess the replacement therapy accurately. Ideally 40-120 mEq of potassium and 4-8 gm calcium along with vitamin supplements are necessary either orally or intravenously. It is also recommended to replace about 350 mEq of sodium per square meter of wound surface in order to maintain the adequate serum levels. If replacement is inadequate not only serum sodium levels fall, even the urinary sodium concentration falls below 40 mEq/l. Once the replacement therapy has been decided upon, the electrolytes have to be checked on a regular basis. The electrolyte imbalance with the use of silver nitrate is noticed mainly in infants and children and in patients with larger burns particularly > 20% TBSA, unless the replacement therapy is maintained. In extremes of age dys-electrolytemia can occur within 6-8 hours of its use and therefore, less frequent saturation of wound dressings is recommended in these age groups.

- The black discoloration of the burn wound surface interferes with further assessment of the wound depth and changes in the burn wound condition.
- Some of the gram-negative bacteria especially *Enterobacter cloacae* metabolize nitrate ion to nitrite which when absorbed through the wound results in methemoglobinemia. Though it is a rare complication, it has to be suspected if there is cyanosis or slate gray discoloration of skin without any pulmonary injury and arterial blood is brown in color even though PaO_2 is normal. Diagnosis can be confirmed by estimating methemoglobin levels in the blood. Management of this condition includes withdrawal of silver nitrate dressing and intravenous administration of reducing agents like methylene blue or ascorbic acid.[2]

The best use of silver nitrate, therefore, is in case the patient is seen within a few hours of burns and silver nitrate therapy is started immediately. If there is a delay of several days and the burn wound has deteriorated because bacterial sepsis has supervened or is impending, silver nitrate will not be effective as it does not penetrate tissues effectively. It can only act on the surface flora. In these cases, other agents with better penetrating ability should be used. Silver nitrate dressings are used in treatment of toxic epidermal necrolysis syndrome and in rare patients allergic to silver sulfadiazine or mafenide.

Mafenide (11.1%)

It is a methylated sulfonamide which was introduced for clinical use as a topical agent for burn wounds in 1964 by Moncrief. It is presently used as a cream with 11.1% concentration of drug in a water miscible base.

Mechanism of action: It is active against most gram-positive organisms and wide spectrum of gram-negative organisms including *Pseudomonas*. It is particularly effective against Clostridial species. It has minimal antifungal action. Its mechanism of action is not known but it is primarily bactericidal only in higher concentrations.

It actively penetrates the burn wound and minimal amount is left on wound surface after 3 hours.[14] After 10 hours, the tissue level is below the concentration required to inhibit bacterial growth. Therefore, to maintain therapeutic levels of the agent in the wound, it has to be reapplied after 12 hours.

After getting absorbed from the wound and entering the circulation, the drug is broken down into its active acid salt which is excreted in the urine. The resultant large osmotic load along with its carbonic anhydrase inhibiting effect results in significant alkaline diuresis. The combined effect c acid load and drug induced renal bicarbonate excr (because of carbonic anhydrase inhibition) leac hyperchloremic metabolic acidosis. This is compens by hyperventilation (to a maximum of 50 L per min This can result in pulmonary complications inclu respiratory failure and pneumonia. These side effects reversible if the drug is discontinued within 24-36 hou

Mode of Application

Mafenide is used as a 11.1% water miscible cream whic applied to wound with a gloved hand in a thickness of mm usually without dressings. As patient is free to mo about in bed, the joint activity is maintained. Usually, tw a day application suffices as a means for controlling bu wound sepsis but reapplication may be required in so areas where the drug gets wiped off the wounds due patient's shifting position. More frequent application unnecessary and may also lead to sulfonamide toxicity whi can be fatal. During reapplication, the remaining cream the wound surface has to be wiped off with wet gau Occlusive dressings are not advocated with the use mafenide because the effective antibacterial action of t drug exists for less than 10 hours only and dressing chan every 12 hours is very cumbersome. Hence, twice a da application by open technique is usually followed.

Advantages

Mafenide is the most useful topical antibacterial agent f control of established or incipient burn wound sepsis whe the bacterial invasion has started or extensive colonizatio is present. This is mainly because it has the best esch penetration ability. An overall reduction in mortality of 50 was noticed with the use of mafenide in a number o studies.[13,17]

Disadvantages

- Application of mafenide is associated with significa pain which is probably because of its high osmolari and hygroscopic effect of the sulfonamide suspensio The painful sensation persists for several minute thereby earning its nickname of 'white lightning' Alternately, applications of mafenide cream can be limit to no more than 20% of the BSA at any one time. Th sites of mafenide applications can then be rotated ever 2 hours until the entire burn has been treated. It is als advisable to alternate mafenide and silver sulfadiazi creams on badly contaminated wounds as a form of 'pulse therapy'.

- 5% of the patients develop maculopapular rash which is characteristic of sulfa sensitivity. It can be easily controlled by oral antihistamines.
- Experiments have shown that its use leads to inhibition of epithelial regeneration also.
- The most significant complication of the use of topical mafenide is the compensated metabolic acidosis which results from its carbonic anhydrase inhibiting effect which decreases the reabsorption of bicarbonate from the renal tubule. The acidic breakdown product of mafenide adds to the acid load. In addition, the bicarbonate is converted to carbon dioxide in the lung and excreted during hyperventilation which is a compensatory mechanism. This is usually seen in patients with > 40 % TBSA burns being treated with mafenide. If the patient is only hyperventilating it may not be a big problem. However, if the patient shows signs of respiratory distress in the form of use of accessory muscles, the drug has to be immediately removed from the burn wound surface and some other topical antimicrobial agent has to be applied. Additionally, ventilatory support and metabolic support in the form of sodium bicarbonate as per the laboratory test reports is also to be provided in these circumstances.
- Patients treated with mafenide also exhibit inappropriate polyuria which results from significant reduction in evaporative water loss from the burn wound (because of 95% water content of the cream base) as well as the osmotic load on the kidney due to the breakdown product of mafenide.
- Hemolytic anemia is a rare complication seen with the use of mafenide.

Despite its disadvantages, mafenide is still the best topical agent for control of established burn wound infection.[18, 19] Its use should be reserved for short duration of time and withdrawn before significant side effects become evident.

Gentamicin[13,20,21]

It is an aminoglycoside used as a 0.1% ointment or cream in burn wound dressing. It is a relatively safe broad spectrum antibiotic.

Mode of Action

It is primarily bactericidal. It is not effective against viruses or fungi. It is effective against both gram-positive and gram-negative organisms particularly *Pseudomonas aeruginosa*. It acts by inducing defective protein synthesis. Indiscriminate use of gentamicin for serious systemic infections has shown development of resistant strains.

Mode of application: It is applied as 0.1% ointment or cream [11]as a thin layer to the wound surface twice a day without dressing or with a light dressing. The application is painless and absorption rate from wound is low. Therefore, the incidence of ototoxicity and nephrotoxicity with topical application is negligible.

It has good eschar penetration but its absorption decreases as the water content of the eschar decreases. Later, as the eschar separates and granulation tissue appears, the rate of absorption of drug again increases. The absorption of the drug is faster if applied as cream base than from ointment base. Therefore, if an occlusive dressing is used, it should be applied as ointment rather than as cream.

Advantages

- Painless and easy application
- Active penetration of eschar
- Low incidence of toxicity when used topically
- Good antipseudomonal action

Disadvantages

- If used indiscriminately, there are high chances of developing resistant strains especially *Pseudomonas*.
- There are chances of ototoxicity and nephrotoxicity with prolonged use.

Although gentamicin gives good results as a topical agent on the burn wound, its use should be reserved exclusively for systemic use.

Bacitracin

Despite lack of efficacy against common gram-negative burn pathogens, bacitracin remains popular in burn care, particularly on partial-thickness face burns. Whether this beneficial effect is due to the antibiotic itself or simply to the bland petroleum base remains controversial.

Less Commonly used Topical Agents

Chlorhexidine Gluconate

Chlorhexidine gluconate has been used in moderate burn injuries either as such or in combination with 0.5% silver nitrate as a prophylactic agent. Its absorption and toxicity are not documented fully and hence, it is not used routinely.

Povidone Iodine

Povidone iodine (Betadine®) has been used as a water miscible base (10%) for burn wound dressing. Though it is found to be effective against gram-negative, gram-positive organisms, fungi, viruses and protozoa, it has little effect on *Staphylococcus strains*. Its use is restricted because of certain disadvantages like its painful application. If used in large quantities in extensive burns, there can be absorption of large quantities of iodine leading to high iodine levels in the blood.

Soframycin, Polymyxin B, Bacitracin

Soframycin, Polymyxin B, Bacitracin have been used for dressing of small burn wounds and are not indicatec extensive burns because of systemic toxicity resulting f absorption of these drugs from the extensive wounds.

Nanocrystalline Silver (Figs 14.23A to E)

In the traditional silver containing antimicrobial agents, s as Silver nitrate and Silver sulfadiazine, most of the sil

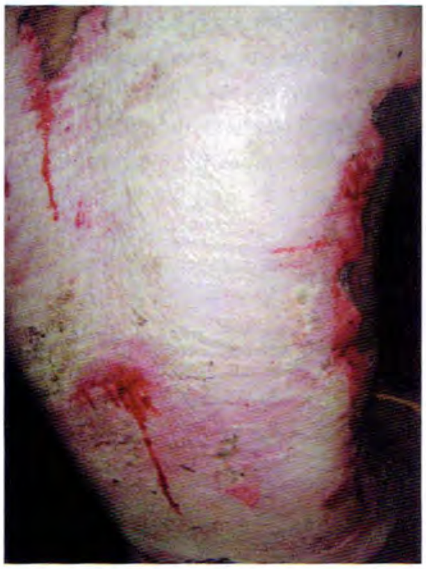


Fig. 14.23A: Superficial to deep dermal burns of the back

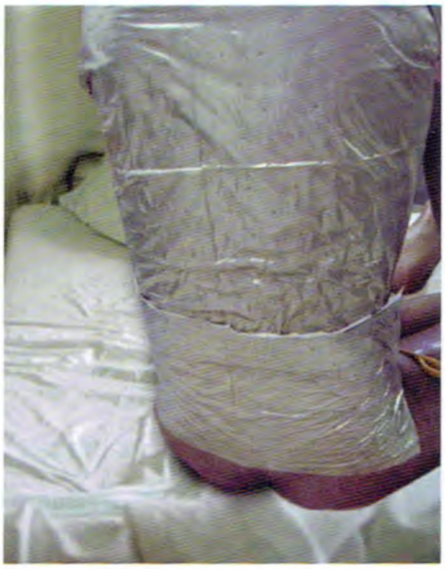


Fig. 14.23B: Nanocrystalline silver dressing (ACTICOAT)® applied to the burns on the back

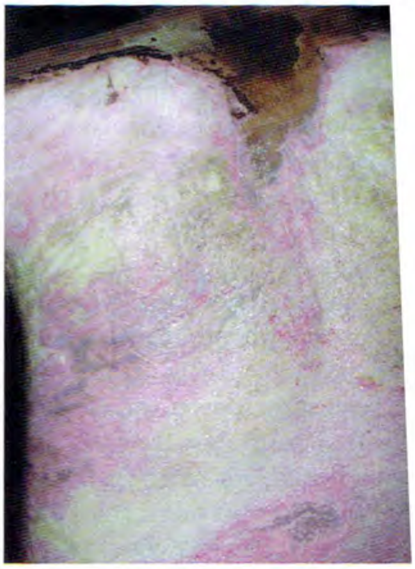


Fig. 14.23C: Wound in healing phase after first dressing with ACTICOAT®

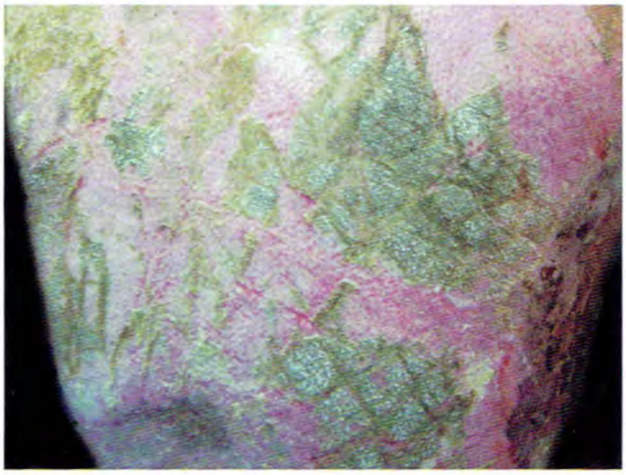


Fig. 14.23D: Wound almost healed after three dressings with ACTICOAT® i.e. after 12 days showing gray staining because of metallic silver

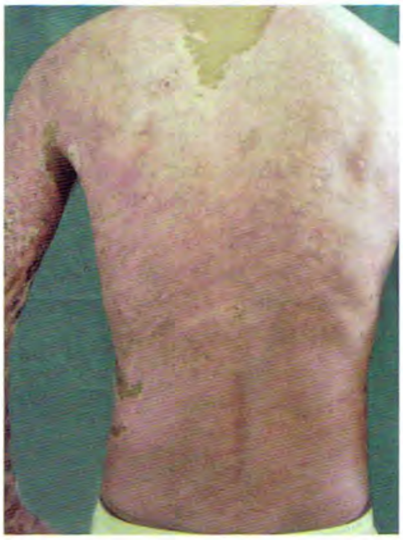


Fig. 14.23E: Same patient after 1 month with well settled scars

carries a strong positive electrical charge which makes it highly soluble, but it also reacts readily with substances such as chloride from the salt in the wound forming silver chloride which is ineffective. As a result most of these compounds allow initial release of silver ions and then most are rapidly inactivated. Therefore it is necessary to apply these agents frequently.

Recently newer products have been launched in the market like ACTICOAT® (Smith and Nephew/Silicryst) which have revolutionized the antimicrobial barrier dressing because they contain nanocrystalline Silver which delivers silver rapidly to the wounds for rapid onset of antimicrobial action to control infection and then gradually over a sustained period of time usually 3 days. As a result the dressing changes are reduced to every 3-4 days. The decreased frequency of dressing also decreases the chances of exposing burn wound to infection from the external environment which is critical to the survival of burn patients.

Another advantage of nanocrystalline silver is that it releases both silver ions and metallic silver. The metallic silver does not react with chloride as quickly as the ionic silver and is therefore not inactivated quickly by wound fluids. Therefore its effect lasts longer without the need for repeated applications.

Summary

Use of topical antimicrobial agents in burn wound care can prevent or minimize burn wound infections and sepsis. In minor and superficial burns, there is no risk for burn wound sepsis and patient may not benefit from the use of topical antimicrobial agents. However, as these cases are treated on an outpatient basis, they are used empirically to prevent conversion of superficial wounds to deep wounds. It is important to understand that any one single topical agent cannot be used in all patients in all circumstances to the exclusion of others. All agents are effective if used in the right circumstances and at the same time, indiscriminate use of any one single agent can be more harmful in the long run. It is therefore imperative to use the topical antimicrobial agents judiciously, selecting the right agent for the right patient at the right time. At times, the drugs can be used in combination in order to reduce their toxicities and overcome their shortcomings.

Thus, the main aim of topical therapy is to control burn wound sepsis and improve the condition of the deep wounds so that they can be closed as early as possible by skin grafting.

FURTHER BURN WOUND CARE

After the initial resuscitative measures and acute burn wound management, the patient comes out of the shock phase and general condition stabilizes. This usually takes about a week in moderate to extensive burns. Now all the attention is paid towards the burn wound as that would then be the greatest threat to the survival of the patient. Apart from the supportive care, the proper wound management per se will help in the spontaneous healing of the partial thickness burn wounds and successful resurfacing of the full thickness wounds.

Modes of Management

After initial dressing, the further wound care varies according to the depth of burn wound. There are various modes of managing burn wounds:

Conservative Treatment

Conservative treatment involves dressings with topical antimicrobial agents only, allowing wounds to heal spontaneously or else reveal granulating wounds which can be split skin grafted. This is mainly indicated for the superficial dermal burns. It may also be indicated in deep dermal and full thickness burns where immediate excision and grafting is not possible. This is usually seen in developing countries where early excision and grafting is not routinely done for all such cases because of paucity of technical support, manpower, postoperative monitoring and overload of burn patients. In these situations, majority of the patients are managed by conservative treatment.

Surgical Management

Surgical management can be:

a. Early excision and grafting
b. Late excision and grafting
c. Progressive debridement and grafting

This is indicated for full thickness or deep dermal burns where the wounds either cannot heal of their own or where they heal with severe hypertrophic scarring or with development of contracture. These surgical techniques are used in different patients or different areas in the same patient. The main aim of treatment is to close the burn wound as soon as possible by the most appropriate method available depending on the specific wounds.

Further care of the burn wound, according to the depth of burns, is as follows:

Superficial Dermal Burns

All partial thickness burns which are superficial dermal heal by proliferation of skin appendages in less than 3 weeks. Therefore, conservative management as described above by either conventional dressings with topical antibacterial creams or biological dressings is normally the rule. The aim of this local treatment is to prevent their conversion into deeper burns by avoiding infection and provide optimal conditions for healing.

Deep Dermal and Full Thickness Burns

In partial thickness burns which are deep dermal, most of the dermis and hence majority of the epidermal appendages have been destroyed. These wounds usually heal after a long time (3-6 weeks) by proliferation of the sparse residual skin appendages residing deep in the dermis after the dead and necrotic tissues come out with conservative treatment. This prolonged period of inflammation and healing leads to hypertrophic scar formation which gives poor aesthetic and functional results. In full thickness burns on the other hand, spontaneous healing of burn wounds is just not possible because of total loss of skin. If left alone, in both these conditions, there are very high chances of septic complications and the surviving patients are left with multiple and severe sequelae as discussed elsewhere in this volume. To overcome these problems, early excision of dead tissue and coverage with split skin autografts should be the standard treatment of choice (Figs 14.24A to 14.25B). A temporary dressing with a good antimicrobial cream needs to be applied while the patient is awaiting surgery. The excisional surgical procedures are described *elsewhere* in this volume.

In patients with very extensive deep dermal burns who may not be fit for early excisional surgery, the routine conventional dressings are continued with intermittent debridement of the eschar till healthy granulation tissue grows and autografting is done. Meanwhile during this waiting period, the general condition of these patients has to be built up continuously as they tend to be in a catabolic phase with negative nitrogen balance till the raw areas are covered. At the same time, one has to be vigilant about patient landing into septic complications during this intervening period. In such circumstances appropriate systemic antibiotics and change of topical antimicrobial agent can save the life of the patient.

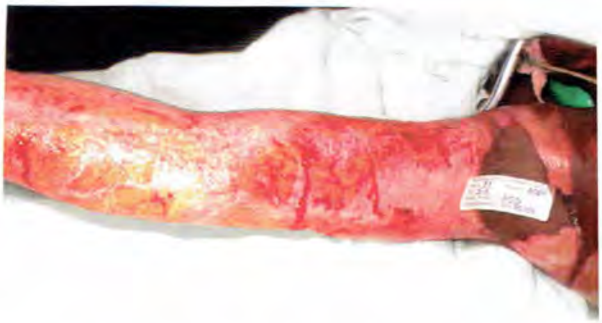


Fig. 14.24A: Early tangential excision of arm

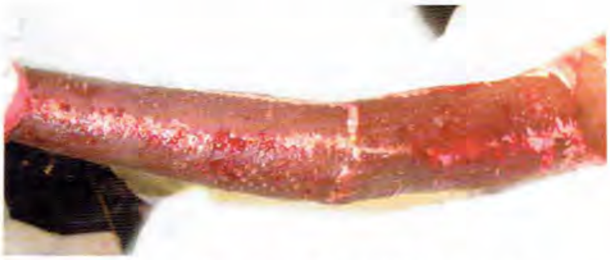


Fig. 14.24B: Split skin grafting of same limb

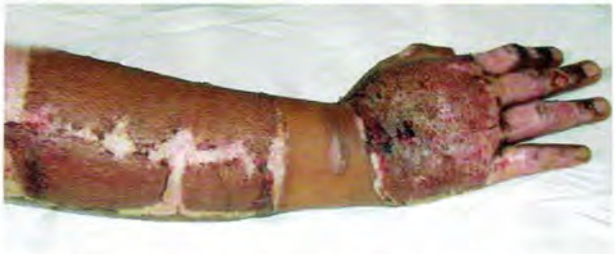


Fig. 14.24C: 7 days postoperative picture of same patient showi good hand movements

Indeterminate Depth of Burns

There are situations where the burn wounds are a mixtu of superficial dermal and deep dermal burns presenting a geographical pattern with some areas showing blanchi together with white non-blanching areas. It is difficult definitely diagnose these areas as superficial or deep parti thickness. Hence, early surgical excision of doubtful are may not be justified as some areas of superficial derm burns may be unnecessarily sacrificed during excisio extending the area of grafting and scarring further to are which would have otherwise healed spontaneously. Th may worsen the final outcome of the healed wounds.

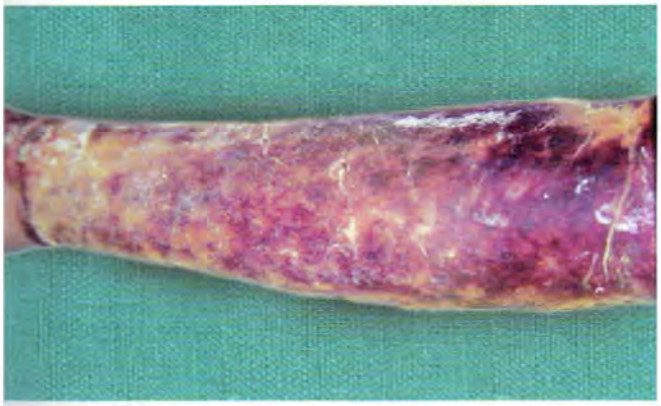


Fig. 14.25A: Dry eschar of full thickness burn on post burn day 10

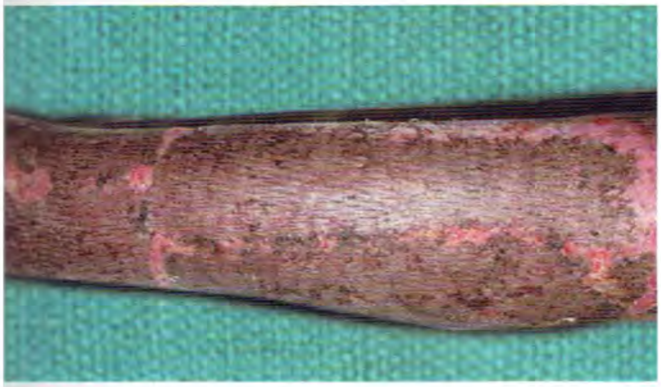


Fig. 14.25B: Split thickness skin graft after excision of dry eschar

These burns have to be treated with an initial conservative approach with conventional dressings for 10-14 days after which reassessment of the area is made. If the wounds appear to be healing and complete wound closure is expected within 21 days, the conservative treatment is continued. Areas which do not appear to be healing are treated as deep dermal wounds and are prepared for delayed excision or debridement and autografting.

Conservative Management

There are different types of dressings used at different stages in conservative burn wound management in order to achieve different goals. The primary dressing in all acute burn wounds have already been described above. This includes open or closed method using topical antimicrobial agent, usually 1% Silver sulfadiazine.

The main aim of dressing is to:

- Decrease evaporative losses
- Prevent and control colonization of wounds by bacteria
- Isolate the burn wound from the other sources of contamination
- Maintain an environment which promotes epithelialization of the partial thickness wounds.

The superficial dermal burns usually heal within 3 weeks with this type of dressing. The first dressing can be left on for 3-5 days provided there is no soakage and smell in the dressing and subsequently at 3 days interval. If there is any soakage, pain, fever and smell, the dressing should be changed immediately and more frequently till the wound condition improves. By three weeks post burn most of the superficial dermal burns heal. It is also easy at this time to identify wounds which are full thickness. For superficial dermal burns, similar dressings can be continued at infrequent intervals till healing is complete. It is the deep dermal and full thickness burns in which there is a lot of slough and the type of dressing needs to be changed as per the requirements of the wound to assist in removal of slough. The non-adherent dressing with silver sulfadiazine or mafenide is continued till loosening of the eschar although separation of slough is slow with use of these antimicrobials. If the eschar is still adherent at 21 days, late surgical excision of eschar can be considered as an option (Figs 14.26A to 14.27B). Another option is **Grid Escharotomy**. In this incisions are given in a grid fashion so as to allow introduction of surface bacteria underneath the adherent eschar and the subsequent pus formation allows the eschar to be lifted off the surface.

Once the eschar is loosened, it is debrided mechanically and the remaining nonviable tissue is best removed by repeated dressings with eusol. This is a wet to dry type of dressing which involves direct application of eusol soaked gauze followed by dry gauze and bandage. Eusol is a good desloughing agent. Eusol (Edinburgh University solution) contains the following:

• Calcium hypochlorite	1 part by weight
• Boric acid	1 part by weight
• Water	80 parts by weight

If patient complains of pain, it can be diluted with normal saline to half or quarter strength. Gauze well soaked in eusol and lightly squeezed out is directly applied to the wound without any paraffin gauze or any other nonadherent layer and covered further by Gamgee pads and bandage. Since this is an adherent dressing, it has to be soaked well with normal saline on removal during dressing change to avoid pain. Ideally eusol dressings should not be allowed to dry for greater efficacy. The action of hypochlorite is transient

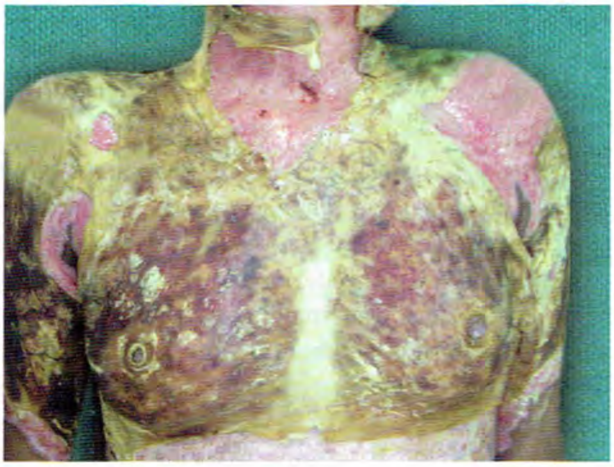


Fig. 14.26A: Traditional wound management—Eschar separation by autolysis

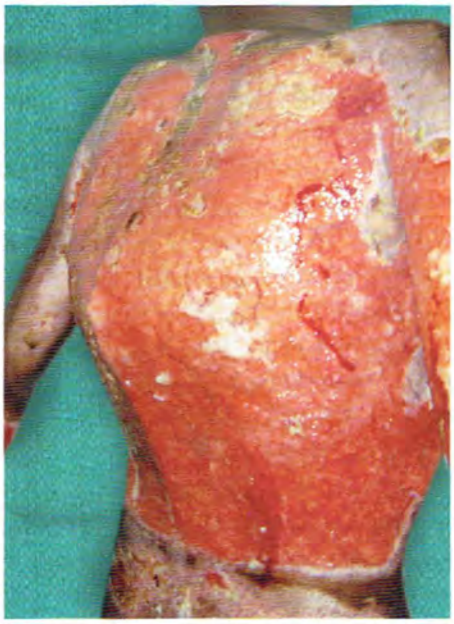


Fig. 14.26B: Granulating burn wound following separation of eschar

so it is advised to repeat the dressing 3-4 times a day. As this is not practicable, the dressing is changed on daily basis. Although the action of eusol has not been studied by any clinical trial, it has been used by surgeons for decades and found to be very effective.

Enzymatic Debridement

Enzymatic debridement using various enzyme preparations (e.g., collagenase –Salutyl®) has been tried from time to time with the assumption that the enzyme can digest the dead tissue and help in earlier removal of esc
Unfortunately, it does not prepare the bed to accept gra
it does not stimulate granulation tissue formation and
even form a neoeschar. As the wounds are not protecte
granulation tissue there are higher chances of inva
infection. In fact reports suggest that patients, whose
wounds are subjected to enzymatic debridement, are
higher risk of sepsis than those treated with to
antimicrobial agents only. Till date none of the enzym
preparations have been found to have much practical val

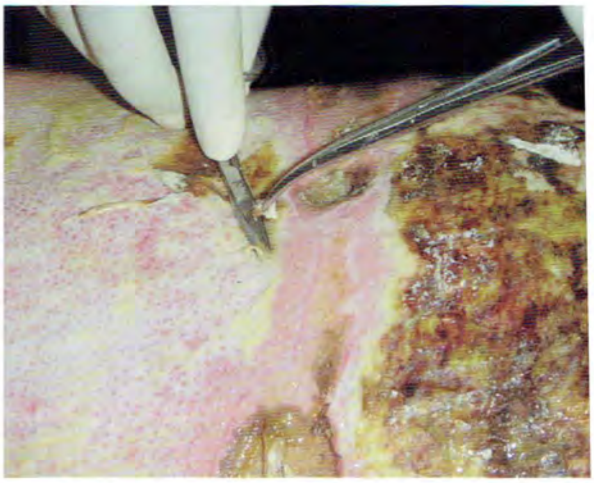


Fig. 14.27A: Preparing wound bed by surgical removal of es

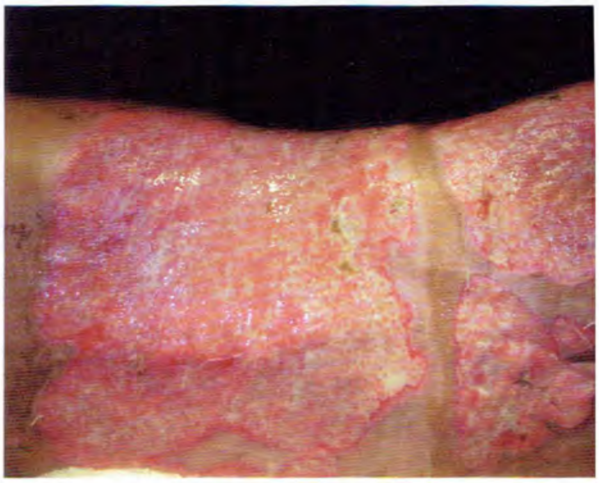


Fig. 14.27B: After eschar separation—The granulation tissue
not healthy – needs further dressing with eusol. Followed by Pov
iodine saline dressing

Once the slough is cleared off the wound surface, an
change in the type of dressing which promotes granul
tissue formation is required. For this simple wet dressi

the form of saline or Povidone iodine lotion gauze is helpful. This includes a layer of paraffin gauze over which saline or Povidone iodine soaked gauze is applied followed by Gamgee and bandage. These gauze compresses have to be in continuous contact with the burn wound and should be wet at all times and replaced preferably twice or thrice a day. As this is not practically feasible, at least once daily dressing is essential. These wet dressings help in development of healthy granulating wounds ready for skin grafting. Wet dressings provide continuous drainage and thus prevent pocketing and abscess formation. These are continued till the patient is fit to be taken up for grafting.

Various growth factors are now being used in clinical trials to hasten wound healing though their efficacy has not yet been fully established. Epidermal growth factor (EGF) [23,24] is one of the recombinant cytokine growth factor which has been successfully used in treating burn wounds. There have been trials of using EGF alone or in combination with silver sulfadiazine cream. Their use has shown marginal improvement in burn wound healing when compared to silver sulfadiazine alone.

SKIN GRAFTING OF BURN WOUND

Skin grafting is the ultimate procedure undertaken during the care of a burn patient and it achieves the final goal of wound closure. The grafts used are split skin grafts taken from unburnt areas of the body. Full thickness grafts have no role to play in post burn raw areas.

History

The history of skin graft dates back to 1869 when Reverdin published his method of skin grafting. He took 1-2 mm diameter skin pieces from a donor area of the patient and transferred it to granulation tissue of skin defect.[25-27] This method was soon introduced throughout Europe despite French surgeons objecting that this method of producing new wounds encourages the risk of erysipelas. Reverdin believed in the principle that the advancing edge of epithelialization makes the wound heal faster. This was also the basis for meshing of the graft. Pollock [28] at St. George's hospital started performing skin grafting in Great Britain in 1870. Reverdin's discovery led to many more clinical experiments which explained the type of granulation tissue best suited for grafting and how the thickness of the skin graft affected the speed of epithelialization.

In 1870, Ash[29] concluded that thinner the graft, the quicker the spread of epithelium around it. With the discovery of the fact that complete coverage of wounds with grafts produced better scars, arose the need for larger grafts. In 1872, Ollier[30], a Frenchman, used grafts as big as 8 cm square.

Gradually surgeons performing eyelid surgery succeeded in covering a wound surface with full thickness skin graft. In 1870, Lawson [31], a British surgeon had applied this technique for upper eyelid reconstruction. In 1875, Wolfe[32] pioneered the full thickness grafts which were then called 'Wolfe grafts' or 'Wolfe Krause ' grafts after Krause gave his experiences with large size full thickness skin grafts in Germany in 1893.[33]

Till this time no importance was given to the wound bed and apart from the patient's own skin, other humans as well as animals were used as donors which led to a lot of disappointing results. The next important criteria for skin grafting were laid down by Thiersch, a German surgeon [34] who on the basis of years of his experience, concluded that grafting should always be done from patient's own skin and grafts should be placed on fresh wound bed and if granulation tissue was present on wound bed it should be excised to improve graft 'take' and lastly, split skin grafts and not the full thickness grafts should be used to cover wound surface.

Thiersch procured grafts using a razor and they were thicker in the middle than at the edges. Therefore, for many years efforts were made to find new ways of improving harvesting techniques in order to get thin grafts of uniform thickness.

Skin grafts were applied only after granulation tissue had appeared, although graft application after excision in case of burns on eyelids on 6th post burn day were also reported.[35,36]

Methods of skin grafting fell into disrepute at the end of the 19th century because of the technique and the disappointing results. Interest in Reverdin's technique was reinvoked at the beginning of the 20th century with modifications. Davis referred to the modified version of Reverdin grafts as 'Small deep grafts'[37] which were later renamed as 'Pinch Grafts' by Gatch.[38] Douglas et al called them 'Steele grafts' after Steele, one of the first British surgeons to apply the skin grafting techniques.

It was in 1929 that Blair and Brown described their successful technique of split skin grafting. They differentiated and elaborated the advantages and disadvantages of thin, intermediate and full thickness grafts. Split skin grafting for burn wounds with their technique became an accepted procedure from 1930 to 1950.The techniques were improved and more importance was given to the preoperative, peroperative and postoperative care. In 1934 Humby formulated various criteria for graft cutting instruments and though his first device did not function too well, he introduced the 'Humby's' knife in 1936 which was a greatly simplified version.[39,40] The original Humby's knife was a razor with a guard which did not allow precise control over the thickness of the grafts. Further modifications were made to increase its reliability.

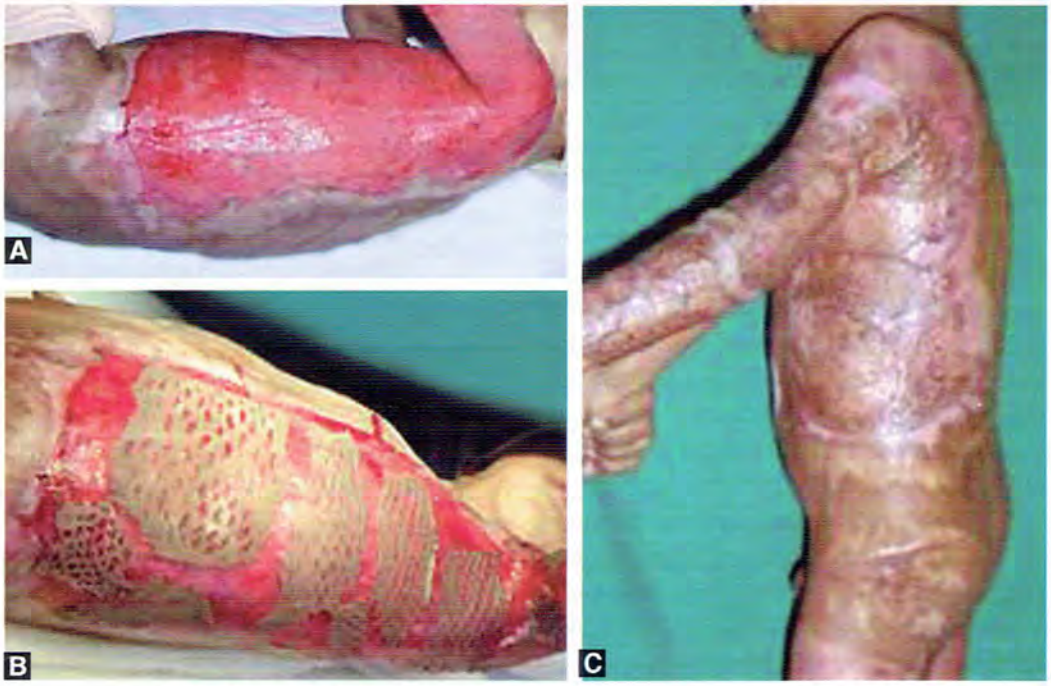
Another instrument which could be adjusted to procure grafts of a uniform width with varying thickness was the 'dermatome'. In 1924, Hagen developed a mechanical dermatome which was driven with a flywheel. In 1939, Earl Padgett, a surgeon and Hood, a mechanical engineer, designed the Padgett-Hood dermatome in USA [41] which could be set at a specific thickness. Brown, another surgeon, developed the 'Brown dermatome' with the help of Barron, a mechanical engineer.[42] This had a rapidly vibrating knife driven by an electric motor.

Preparation of Wound for Grafting

A couple of days prior to grafting, the paraffin gauze is omitted from the dressing so as to make the raw surface non-greasy for better adherence of skin graft.

The condition of the granulation tissue is quite sugg
of the wound preparation for grafting. A healthy bed w
is ready for grafting shows clean red granulations d
of any necrotic areas and does not bleed readily on touch
14.28). If the wound looks angry red and granulation t
is very fragile and bleeds readily on touch with unhe
copious discharge, it is suggestive of beta-hemo
Streptococcus infection (Fig. 14.29). This is the only
contraindication for skin grafting of wound. S
Streptococcus is still found to be sensitive to penicilli
other antibiotics, systemic penicillin should be st
immediately and continued till swab culture is negativ
this bacterium. If the granulation tissue appears slimy
suggestive of *Staphylococcus* infection (Fig. 14.30). In
cases, systemic antibiotics, based on wound swab cul
sensitivity data are administered and local dressings are
with anti-staphylococcal agents like fucidin, mupirocin
Skin grafting is done only after *Staph.aureus* is cleare

If there is edematous, hypertrophic granulation ti
gentle compressive dressings using crepe bandages s
be done to reduce edema. Local steroid cream is
effective (Fig. 14.31). Bluish green pus discharge fro
wound suggests *Pseudomonas* growth (Fig. 14.32). In
cases, dressing with 1% acetic acid solution is very effe
or else mafenide can be used (Fig. 14.33). Pale granul



Figs 14.28A to C: Healthy granulating wounds covered with split skin graft

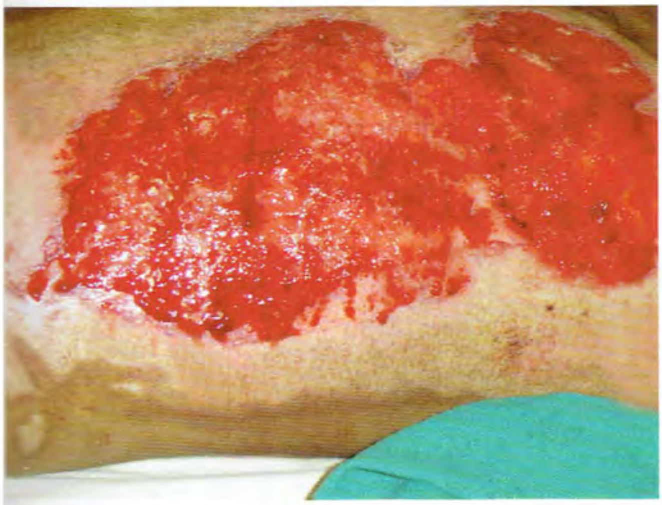


Fig. 14.29: Fragile Granulation tissue bleeding profusely on touch – Indicating streptococcal infection

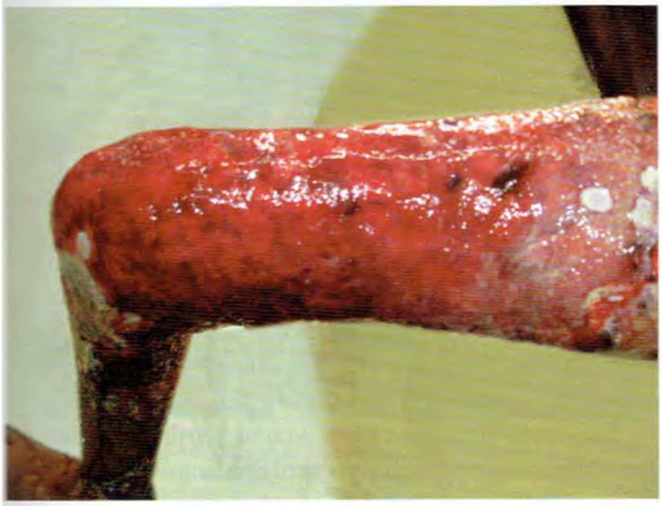


Fig. 14.30: Slimy dirty granulation tissue with copious discharge Indicating *Staphylococcus* infection

tissue suggests anemia which should be corrected by blood transfusions (Figs 14.34 and 35).

Once granulation tissue appears, swab cultures are always taken to look for microbial growth and its sensitivity pattern to various antibiotics before taking up the patient for skin grafting. Growth of beta-hemolytic *Streptococcus* is an absolute contraindication for grafting. Initial 'take' of graft followed by loss after a few days with an eaten up appearance is typically indicative of *Staphylococcus aureus* infection (Fig. 14.36).To prevent further loss of graft, action has to be taken immediately as advised above. Sometimes the burn wounds are neglected and they turn into chronic

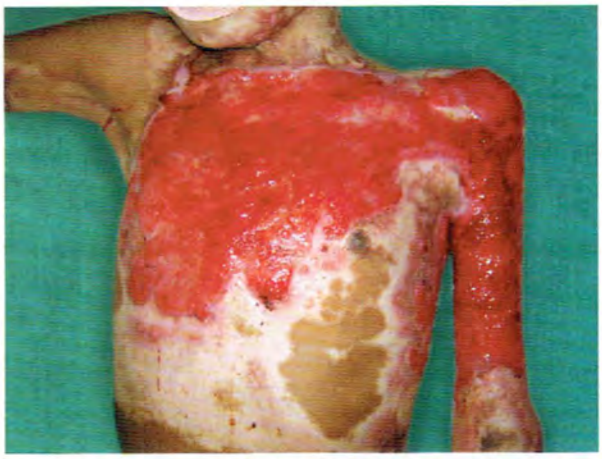


Fig. 14.31: Edematous hypertrophic granulation unfit for grafting

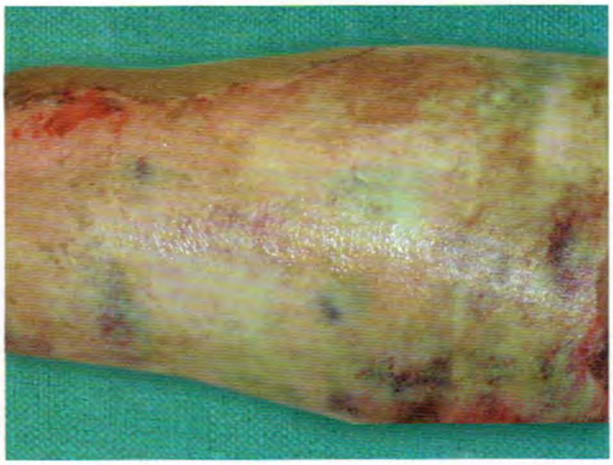


Fig. 14.32: Greenish discoloration of wound indicative of *Pseudomonas* infection

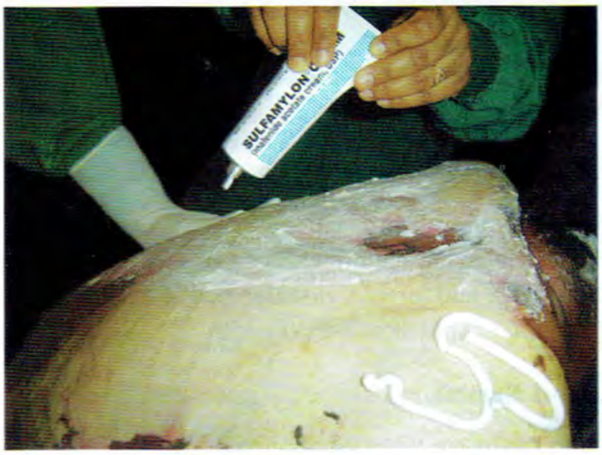


Fig. 14.33: Dressing with mafenide or sulfamylon cream to counter *Pseudomonas* growth

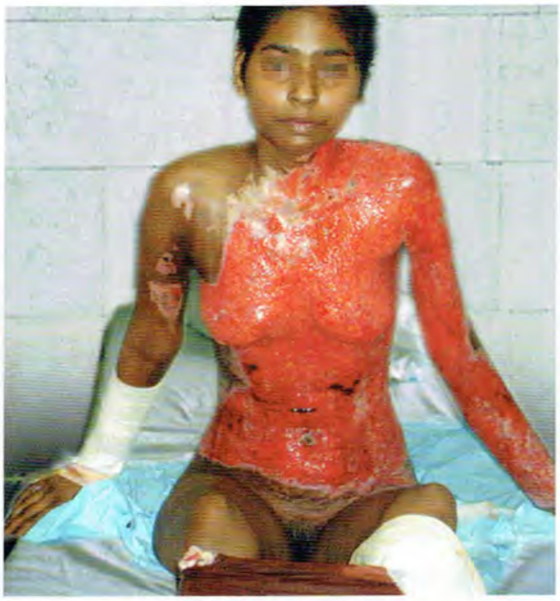


Fig. 14.34: Severely pale wound with scarce granulation, at places underlying fat is also visible

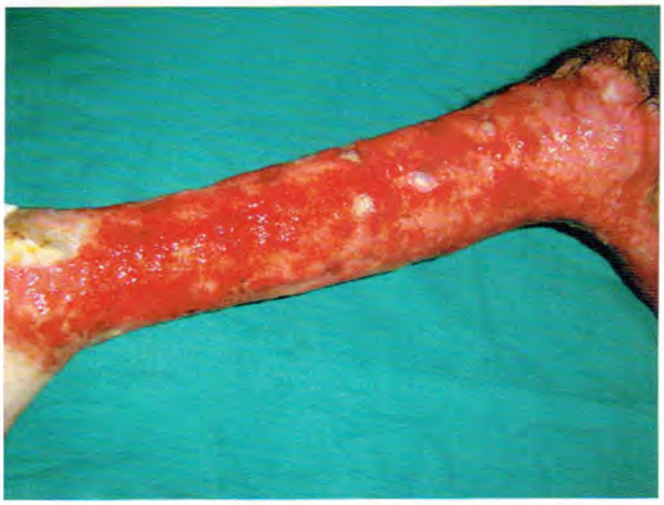


Fig. 14.35: Old burn raw area with fibrosis occurring in the bed with pale and very thin granulation tissue

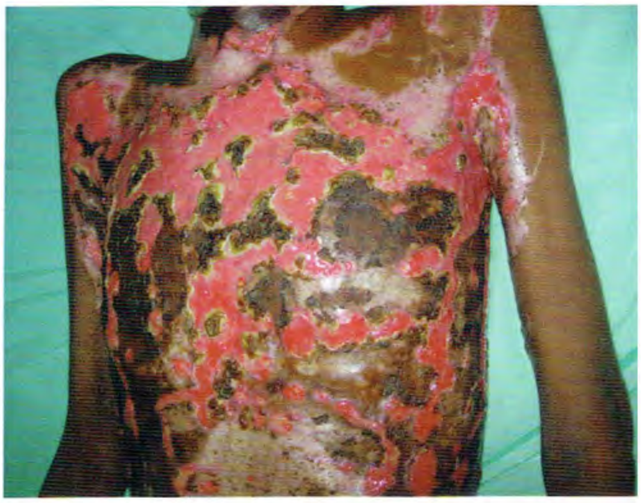


Fig. 14.36: Loss of skin grafts with MRSA infection

fibrosed wounds.These wounds cannot be prepared by dressing alone and require excision of the fibrotic bed till healthy soft and supple vascular bed is reached before application of split skin graft (Figs 14.37A and C).

Wound Swabs

Wound swabs are taken on admission to know the state of wound at start of treatment and then on first dressing, i.e. third or fourth day and thereafter once a week till wounds heal. Wound swabs can be repeated more often to kno the change in bacterial flora of the wound and its sensitivi pattern if any unexpected worsening in condition of th patient and/or wound occurs. When the patient is taken u for grafting, wound swab is sent 3 days prior to surger for culture and sensitivity examination.

Local antibiotic agents have a poor effect in controllin local burn wound colonization. At the same time, if dea tissue is present both local and systemic antibiotics are n very effective because they cannot enter the dead tissue. I such circumstances, the dead tissue or slough has to b debrided rapidly and then both local and systemic antibiotic as per the sensitivity report, are started.

For cases where the burn wound sepsis is caused b *Pseudomonas aeruginosa*, frequent dressings with mafenid is very effective as it penetrates eschar very well and clea the organism within a week. Since the separation of sloug may be delayed by the use of mafenide, surgical excision o dead tissue may be required.

Usually when the slough separates there is a layer o granulation tissue already forming under it, though it ma not be very healthy. Slough separation can be hastened b using wet to dry dressings which help to soften the escha and since they are adherent to the eschar when the dressin begins to dry, they help in removal of slough from the woun surface during change of dressings.

In case of deep dermal or deep partial thickness burn the wounds are inspected at regular intervals to look fo surviving epithelial elements which can produce islands o regenerating epithelium which spread and then coalesce t heal the wound completely. A smooth white bed of derma

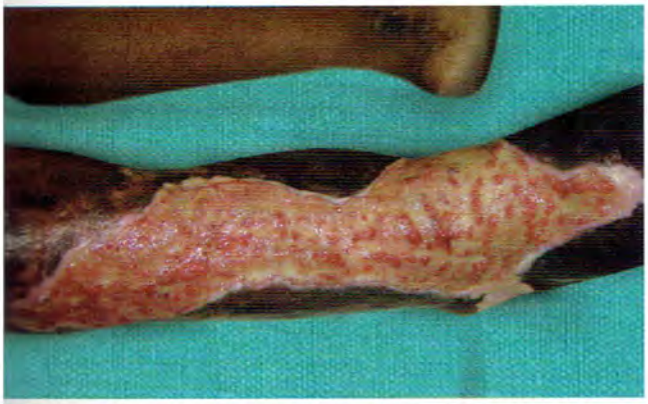


Fig. 14.37A: Old neglected post burn raw area with fibrosed bed with slough and no granulation. This wound needs to be excised to remove the unhealthy tissue

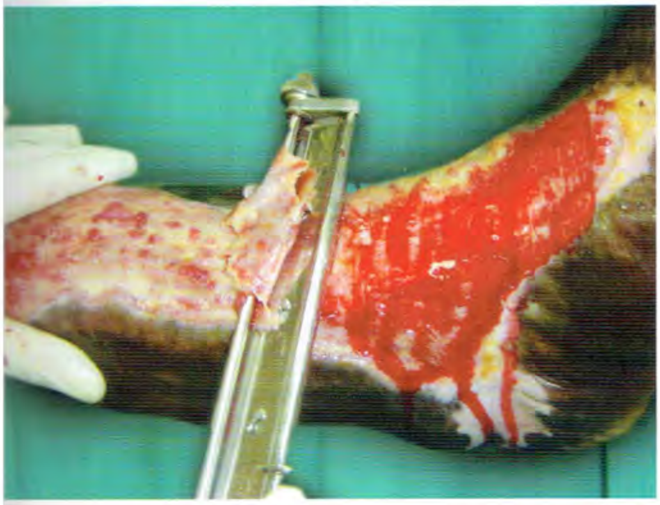


Fig. 14.37B: Excision of the same wound

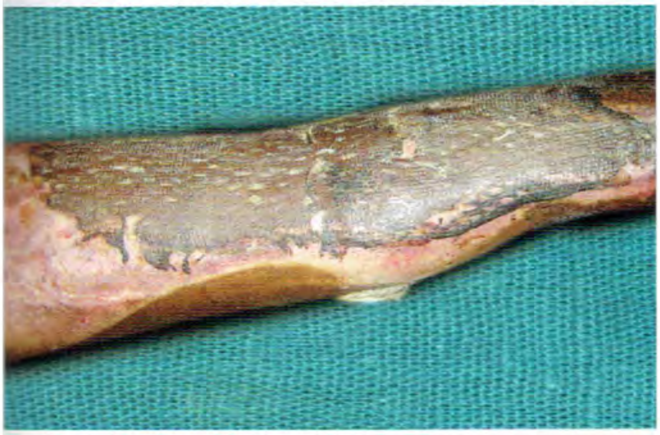


Fig. 14.37C: 10 days after application of skin graft which is fully taken up on the healthy bed post excision

collagen intermixed with red islands of granulation tissue usually indicates a deep dermal burn with enough epithelial elements which can heal the wounds although very slowly and hence with a lot of hypertrophy.

Types of Skin Grafts

There are different criteria for classifying skin grafts.

Based on the Anatomy

Based on the anatomy skin grafts can be classified as follows:

(a) Partial thickness or Split thickness skin graft (SSG) – It includes epidermis and part of dermis. Though they can survive on less vascularity from the bed, they have greater tendency for contraction. These are most effective for post burn raw area coverage. Donor sites of split thickness grafts heal spontaneously by re-epithelialization from epithelial cells lining the hair follicles and sweat glands within 10-21 days. These can be reharvested once the wound epithelializes.

(b) Full thickness grafts (FTG) –Include epidermis with entire dermis and are approximately 0.035 to 0.045 inch thick. They require better vascular bed for survival as well as good vascularity from the margins. They undergo minimal contraction. Their sensory recovery is better than that of split skin graft. They have no role to play in post burn raw areas but have an important role to play in covering areas following post burn contracture release. The donor areas for full thickness grafts cannot resurface spontaneously because of lack of epithelial elements. The defect needs to be closed primarily. Therefore, they are not used for large defects.

The post graft contracture is actually due to contraction of the wound bed. It is widely believed that thicker the graft, greater will be the inhibition of contraction. This holds good only if the grafts are harvested from a given site. The fact is that when split-skin grafts are harvested from different sites it is the total percentage of dermal thickness grafted, which determines how much contraction will be inhibited. Delayed application of skin graft does not inhibit contraction as effectively as immediate grafting.

Based on Thickness

Based on thickness the split thickness grafts can be further categorized into 3 types:

a. Thin split thickness graft—which has a thickness of 0.006 to 0.012 inch.

b. Intermediate thickness graft—which has a thickness of 0.012 to 0.018 inch

c. Thick split thickness graft—has a thickness of 0.018 to 0.030 inch.

Post burn raw areas should be covered by thin and intermediate thickness split skin grafts.

For treating burn patients with skin grafts, apart from the age and sex there are many more factors to be considered when deciding the thickness of the graft to be harvested. By and large, for all burn wounds, grafts should be thin because their 'take' is better. However, because they undergo greater contraction, thicker grafts (intermediate thickness) are preferred for regions like face especially eyelids and lips so that they contract less postoperatively. In children and elderly, usually thin grafts are harvested as the dermis is thin. If a donor site is to be reused more than once for procuring grafts as in patients with extensive burns, thin grafts should be harvested. This is because the donor sites may not heal due to loss of dermis if thick split thickness grafts are harvested repeatedly.

Based on Size and Technique

Based on size and technique of harvesting split skin grafts they are classified into following types;

(a) *Pinch grafts*—These are 'small deep 'grafts' which are not used at present because of the extremely ugly scarring of the donor site. The donor area chosen, therefore, is a hidden area usually the thigh or groin. The procedure involves lifting the skin with a needle and a 0.5 cm fragment is cut to obtain a graft which is thicker in the center and tapering towards the edges. The resulting wounds can even be closed primarily. The grafts are transferred to the wound at regular intervals, distance between them being about 5 mm. This method is now of historical importance only.

(b) *Stamp grafts* – These are split thickness grafts which, after harvesting, are cut as square or rectangular pieces of skin of different sizes. They are applied to the wound and held in place with occlusive dressings. The intervening areas between the stamps heal by epithelialization from the margins of the graft. As a result, these areas tend to hypertrophy and give unsightly scars. This method may be of use in case of very extensive raw areas.

(c) *Sheet grafting* – It is the most widely used at present as the grafts can now be easily harvested with the help of the latest modified Humby skin grafting knife or dermatome. Large sheets about 10 cm wide or more can be procured easily and they can cover large raw areas. This gives good aesthetic results especially on the face. These sheets meshed to increase their size to cover larger areas.

(*d*) *Meek grafting*– It is being used lately. This was des in 1958 by an American surgeon, CP Meek. It invol use of a 'Meek-Wall ' dermatome to obtain postage autografts which are expanded using double p gauzes.This produced a regular distribution of aut islands with a ninefold expansion but required donor of suitable size and shape.It was discontinued for a after the introduction of Mesh grafts in 1964, beca problems encountered with the old technique of grafting. The meek grafting technique was redesign overcome the imperfections of the original method. A s spray called 'meek adhesive' was developed an prefolded gauzes were manufactured with expansion of 1:3, 1:4, 1:6 and 1:9. This required very small areas and the graft islands were evenly placed in a re pattern. The epithelialization and hospitalization ti reported to be shorter with this technique, compared mesh graft method. This method is preferred in exte burns where the donor areas are very limited.[52,53]

Indications for Skin Grafting

Skin grafting is indicated in the following situations in b

1. Following excisional surgery of burn wounds
2. Post burn raw areas following full thickness burns
3. Following release of post burn contractures
4. Following excision of post burn scars like hypertro or hypopigmented scars, etc.

Essential Criteria for Skin Grafting

1. Grafting of post burn raw areas should be done as s as the granulation tissue is healthy. If delayed for lo the granulating areas become fibrotic and graft 'take less than satisfactory.
2. The granulation tissue should have healthy appearance i.e. it should be a well vascularized bed.
3. The wound bed should be free from infection. Wou cultures should always be done prior to grafting. T presence of beta–hemolytic *Streptococcus pyogenes* an absolute contraindication for grafting as it does allow the graft to 'take' at all as it eats the fibrinog which is responsible for graft adhesion to the b Presence of other bacteria like *Pseudomonas aerugino E. coli, Klebsiella* is not a contraindication for grafti if present in < 10^5 organisms/gm of tissue. They hinder graft 'take', yet not to the extent as to del

grafting. Although *Staphylococcus aureus* allows graft to 'take' initially, it eats away the graft progressively to unacceptable degree and it is not advisable to graft a wound until it has been eradicated. In extensive raw areas it is not possible to have bacteria-free surface.

4. The general condition of the patient should be stable. Anemia should have been corrected by blood transfusion(s). Hemoglobin should be a minimum of 10 gm%. The patient should be built up nutritionally (both enteral and parenteral). By and large, the patient should be stable enough to bear the stress of surgery.
5. Quantitative culture of the wound should be sent if infection is suspected. Counts of $>10^5$ organisms /gm of tissue should be first treated using systemic and topical antimicrobial therapy and frequent change of dressing till the colony count decreases.
6. Complete hemostasis should be achieved in the wound bed prior to graft application especially if the granulations have been scraped. This can be achieved by pressure dressing with adrenaline soaked gauze or spray of fibrin glue (Tissel®). However, if there are many areas of adherent slough in the granulation tissue which cannot be rubbed off using dry gauze, they should be tangentially excised with a scalpel or skin grafting knife followed by pressure dressing with adrenaline packs.
7. Granulation tissue, if unhealthy, can be removed or scraped from the wound bed. However, it causes more bleeding which impairs graft 'take' if applied immediately.
8. Immobilization of the part without shearing between the graft and bed is very essential.

Donor Areas

Skin grafts can be harvested from any area of the body including scalp and feet if there is scarcity of donor areas. The most common sites whenever available are the lower limbs and upper limbs.The thigh is the most commonly used in lower limb especially the anteromedial and anterolateral thigh. (Fig. 14.38) The back and buttocks are useful sites if thighs are also burned. If the limbs and back is burnt, the chest, abdomen or scalp can be used as donor areas. In these areas, adrenaline saline (1 in 2,00,000) can be injected subcutaneously to eliminate the depressions of the intercostal areas or the curvatures of the abdominal wall or bony prominences in order to form a flat base from which it becomes easier to harvest a good sheet of graft. It also reduces blood loss from donor site.

Similarly, scalp is shaved and infiltrated with adrenaline saline solution before harvesting the graft. The advantage with scalp is that grafts can be harvested repeatedly, every 10 days, as epithelialization is very fast from the epidermal lining of the large number of hair follicles (Figs 14.39A and B).

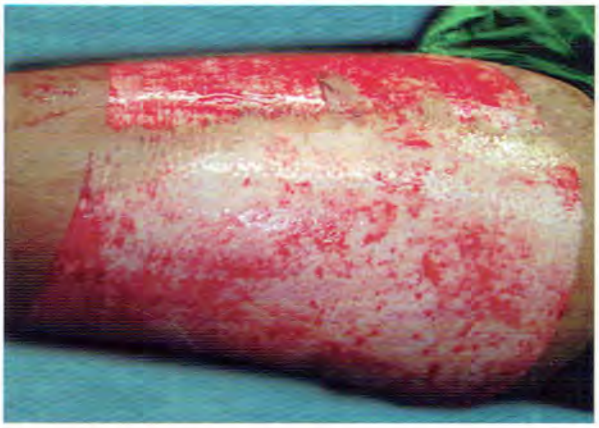


Fig. 14.38: Anterolateral and anteromedial aspect of thigh–Commonest site for harvesting graft

While harvesting grafts from a hairy region, it is important to take thin grafts as thicker grafts contain undesirable hair follicles which lead to hair growth in the recipient area.

The donor areas of split skin graft heal in 10-21 days. The donor areas are dressed by application of paraffin gauze which may or may not be antibiotic impregnated followed by gauze and then Gamgee pads and a compressive bandage. This bandage usually gets loosened up in 2 weeks and gauze falls off when opened in 3 weeks as majority of the superficial donor area re-epithelializes by 12 days. The other dressings which have been used for donor areas include collagen dressings, Biobrane and Opsite. All these have been found to reduce pain, act as a barrier to bacteria, fasten healing and reduce the bulk of dressing. However, they are very costly.

Instruments for Grafting

There are various skin grafting knives available and they have been modified from time to time to improve the quality of graft which can be harvested with their help. The earliest free-hand instrument to be used for grafting was the *Blair knife* which required manual sharpening regularly. Then came the *Humby knife* which had a fixed blade but an adjustable roller guard which adjusted the thickness of graft harvested. The amount of pressure applied to the instrument on the surface of the skin also altered the thickness of the graft, i.e. the greater the pressure applied, the thicker the

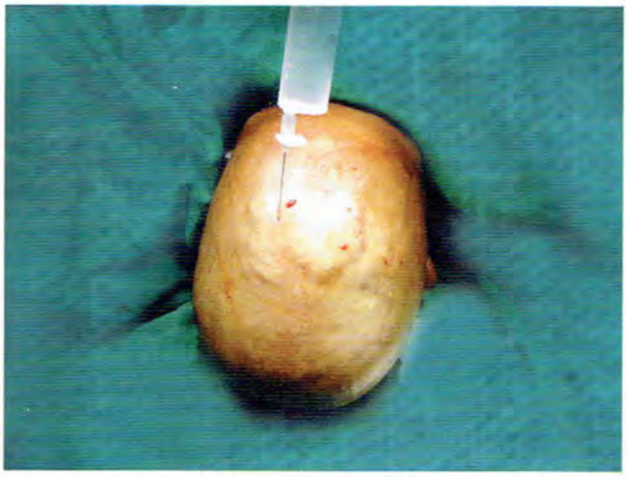


Fig. 14.39A: Scalp as donor area – Saline infiltration being done

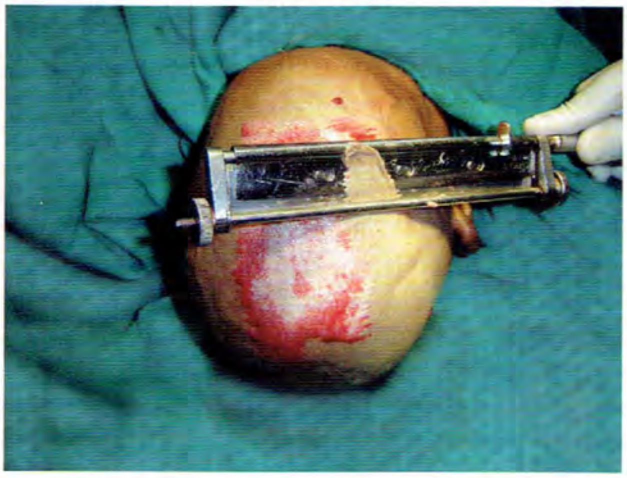


Fig. 14.39B: Graft being harvested from scalp

graft and lesser the pressure, thinner the graft. There were various modifications to the Humby knife with disposable blade, e.g. *Braithwaite knife*–in which the roller guard moved longitudinally as well as rotated on its axis so that the graft tends to roll on to the rod and its edges were ragged. Then came another modification—*Bodenheim knife*– in which the roller guard moved only to and fro but did not rotate on its axis.The latest is the *Watson's modification* of Humby Skin Grafting Knife (Fig. 14.40A) in which the guard is fixed and immobilized so that the edges of the graft are clean cut rather than ragged and the graft does not roll on to the rod as the knife proceeds forward. This is the most commonly used manual skin grafting knife at present. For procuring smaller sized grafts, 'Silver' knife has been used for many years (Fig. 14.40B).

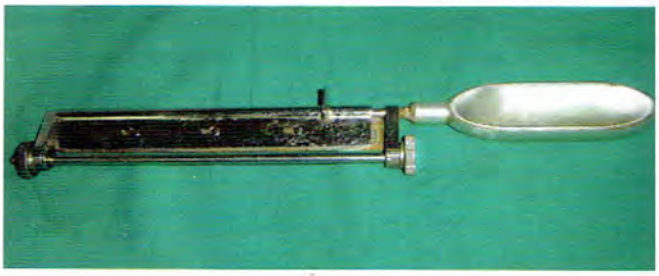


Fig. 14.40A: Watson's modification of Humby's knife—Most commonly used manual skin grafting knife

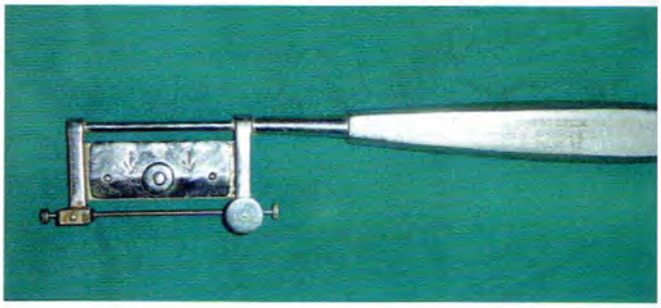


Fig. 14.40B: Silver knife—Used for harvesting small grafts

The adjustment of the guard for varying the thickness of the graft in Watson's skin grafting knife is done by the screws present at both ends of the roller guard according to the calibrations present on proximal screw and then tightening the distal one. Another method for assessing then required gap between the blade and the guard is by holding the knife against the light so that the gap between roller guard and the disposable blade can be seen and the screws tightened. Increasing or decreasing the distance between guard and the blade produces thicker or thinner grafts respectively. Even increasing the angle at which the knife is kept on the surface of skin and the pressure exerted varies the thickness of graft harvested.

The free hand knives have been replaced by dermatomes (Figs 14.41A to D). *Padgett-Hood dermatome* was a mechanical drum dermatome in which the depth of cut could not be modified. After this the *Brown dermatome* was introduced which was driven by an electric motor. The advantage with these motorized dermatomes was that they produced uniform long grafts with predetermined width and depth. The *air Zimmer dermatome* powered by compressed, water pumped, nitrogen was introduced and was easier to use. Dermatomes may not be useful when the donor areas are narrow.

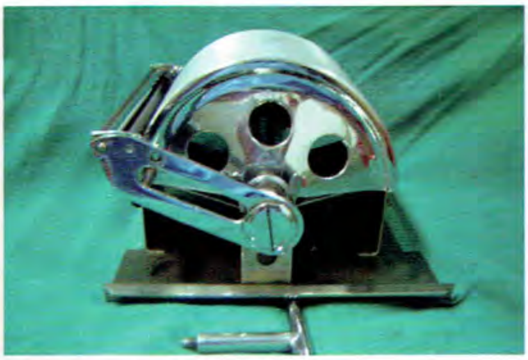


Fig. 14.41A: Padgett's drum dermatome

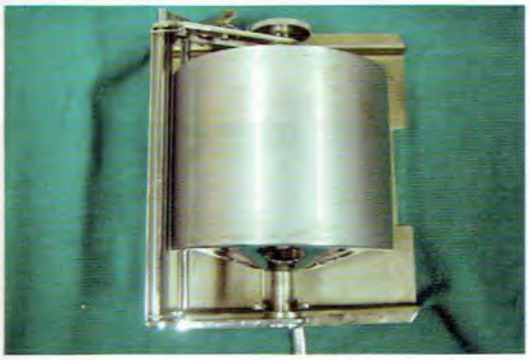


Fig. 14.41B: Same dermatome showing the surface of the drum on which the glue is applied when placed in contact with the skin

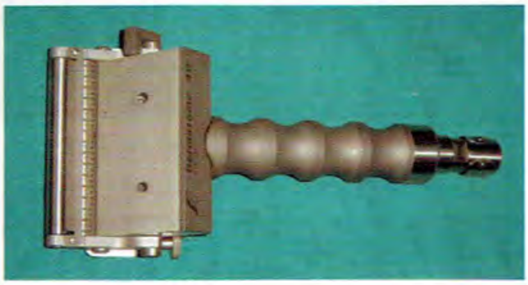


Fig. 14.41C: Electric dermatome

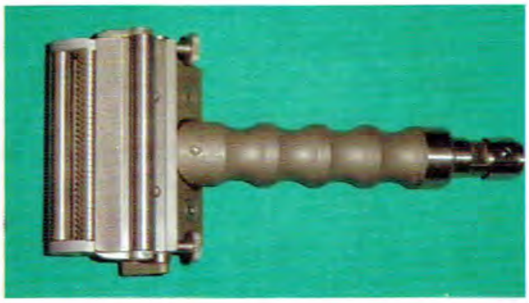


Fig. 14.41D: Electric dermatome–Another view

Technique of Harvesting Split Skin Graft (Figs 14.42A to D)

Once the donor site has been selected, the graft can be harvested under general anesthesia, regional nerve blocks or even local anesthesia. Infiltration of local anesthesia with adrenaline reduces the amount of blood loss from the donor area apart from prolonging the duration of the effect of the local anesthesia. The area is lubricated with Vaseline or paraffin and saline. An assistant gives traction to the skin from one end of the donor site while the operating surgeon gives traction from the opposite side. Wooden / metallic Gabbroo graft boards are frequently used for applying the traction and counter traction. The knife is placed parallel to the surface of skin and then moved 'to and fro' with moderate pressure and gradually moved ahead with moderate speed till a long sheet is removed. The width of the graft harvested can be adjusted according to the area available.

In case of dermatome, it is kept on the skin surface at an angle of 30-45° and the throttle is pressed and the dermatome advanced flat to the skin with gentle downward pressure. Once the sheet of graft is harvested, the dermatome is angled upwards and lifted off the donor site cutting the graft.

Thin grafts can also be taken from areas adjacent to burn if other donor sites are not available. There is no risk of infecting the donor areas. Whenever the raw areas are extensive and donor areas limited, the split skin graft can be meshed to expand the graft to cover the whole defect.

Meshing of Graft (Figs 14.43A to 14.44C)

Meshing of a skin graft is an old technique first described by Lanz of Amsterdam in 1907[46] when it was done manually. It was re-introduced by Tanner, Vandeput and Olley(1964) with a new instrument.[43] They used a device that made slits in the graft at regular intervals producing a net like structure when the graft is stretched. Tanner et al were inspired by Dragstedt and Wilson (1937)[44] who used the same technique in full thickness graft for drainage of fluid from wound bed.They, in turn, were impressed with the 'seive' graft developed by Douglas (1930)[45] in which circular holes were cut in a full thickness graft by leaving small islands of skin on the donor site for re-epithelialization.

The principle behind meshing a graft is to gain expansion by making incisions parallel to the longitudinal axis of the graft and the maximum advantage of meshing is gained when the long axis of the meshes parallel the stress lines of the area to be grafted.

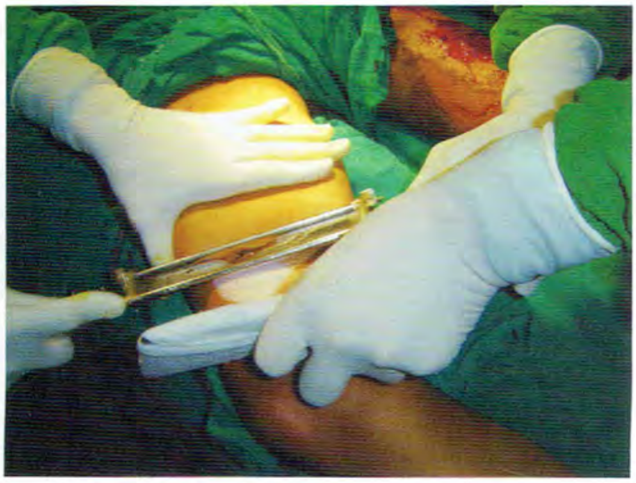


Fig. 14.42A: Technique for harvesting skin graft with Watson's knife

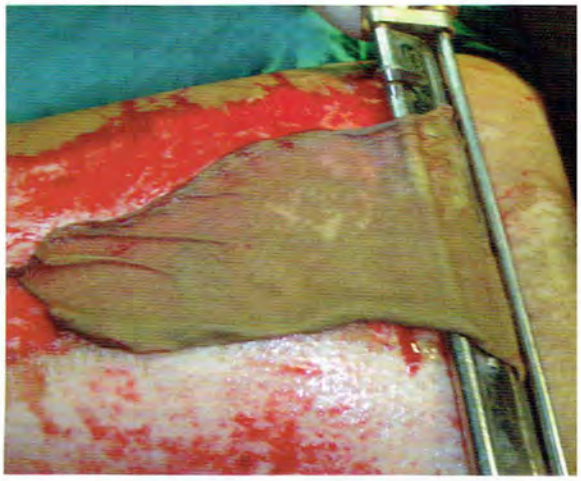


Fig. 14.42B: Thin split thickness skin graft

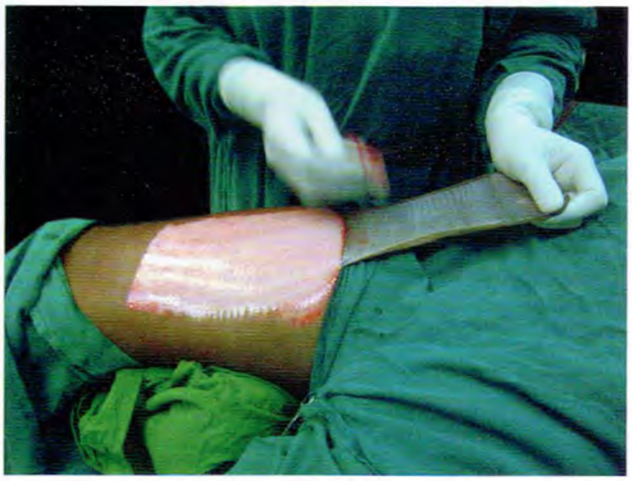


Fig. 14.42C: Intermediate thickness skin graft

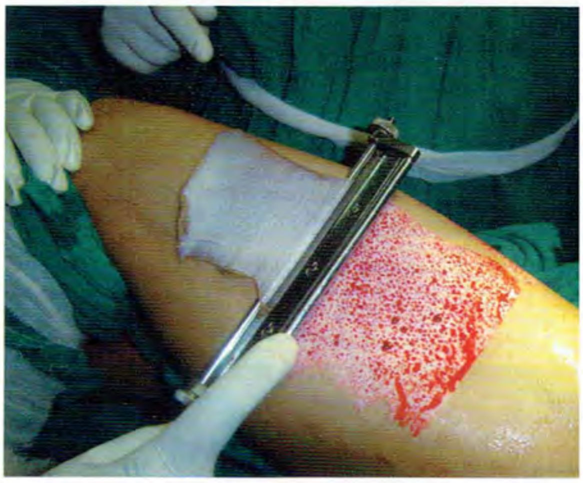


Fig. 14.42D: Thick split thickness skin graft

Earlier, incisions in graft were made using a roller which was rolled over a bed of small blades. Later the graft was placed on a plastic carrier with slanting grooves and both the carrier and graft was drawn through a sort of mill with revolving blades. The technique has been steadily improved with modifications added to the older meshing instruments like Padgett's mesher. The latest is the Zimmer's mesher in which the carrier with the graft can be pulled through the mesher with the help of a handle or lever. By adjusting the lengths of the cuts, the resulting size of the graft can be changed and expansion of 1.5 to 9 times the original size can be gained. Minimal expansion should be used depending on the availability of the skin graft in relation to the size of the raw areas.

In order to get good expansion by meshing, the grafts should not be too thin otherwise they tend to get torn. The successful use of mesh grafts in covering extensi
areas has proved its worth.

Advantages

The advantages of meshing are as follows:

1. It expands the graft.
2. Discharge can escape through the gaps in the g
that the accumulated discharge does not lift th
off the wound surface.
3. The meshing gives a larger edge for production
epithelial cells than the stamp grafts or strip gr
meshed graft conforms better to the wound s
When the slits are made, the edges of the sli
towards the dermal surface because of the in
epidermis and elastic dermal layer. When this r
applied to the raw area, both undersurface as

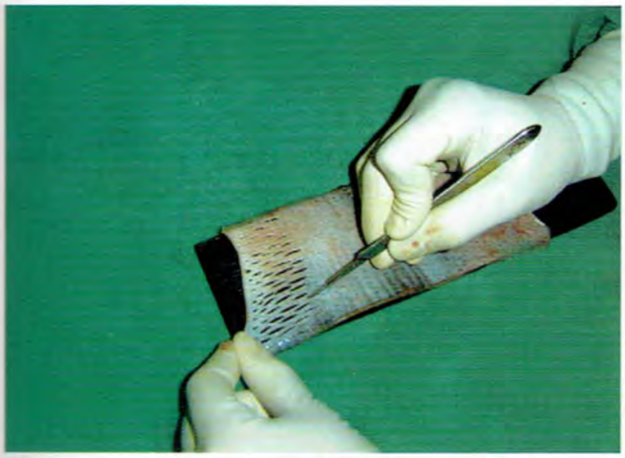


Fig. 14.43A: Meshing of graft being done by hand

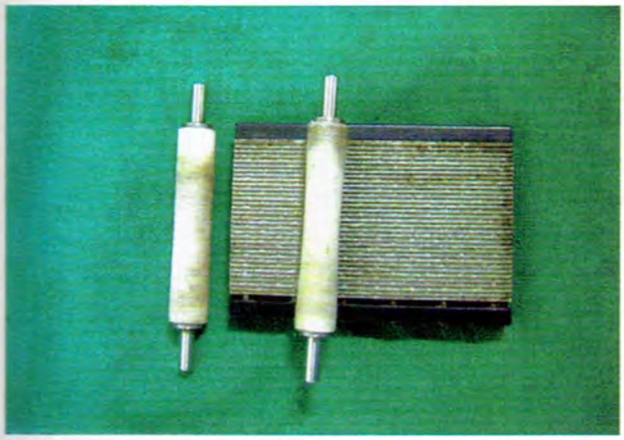


Fig. 14.43B: Padgett's roller skin graft mesher

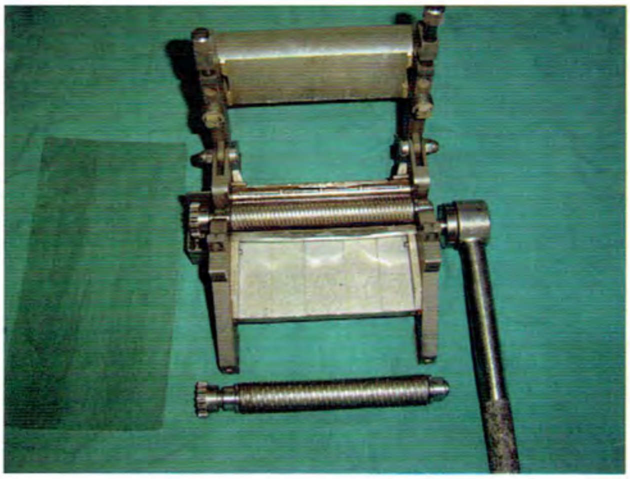


Fig. 14.44A: Zimmer's skin graft mesher

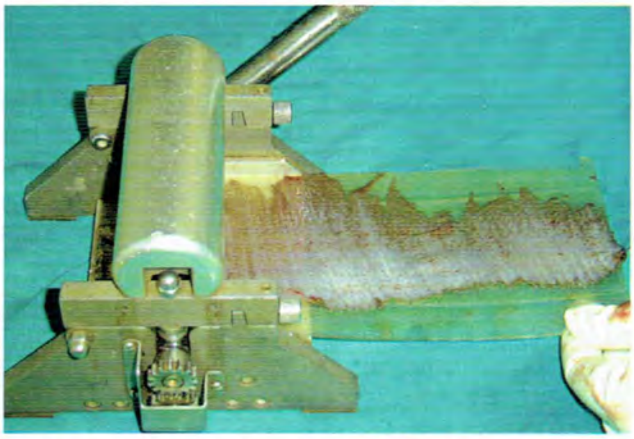


Fig. 14.44B: Graft being meshed by passing it through the mesher

the edges of the skin bridges adhere to the recipient bed. This allows for greater total contact of graft to the wound.

4. The raw areas seen within the mesh heals by the epithelium migrating from the slit margins and this takes only a few days as the raw areas within the mesh are always less than 5 mm wide. It is well known that epithelium spreads rapidly from the edge of the defect for a distance of 5 mm and thereafter it spreads very slowly.
5. Meshed graft can be easily placed in close proximity to the wound surface in case of uneven or convoluted surfaces.
6. It is easier to express out a hematoma or seroma from under the surface of the graft without disrupting the graft if it is meshed.

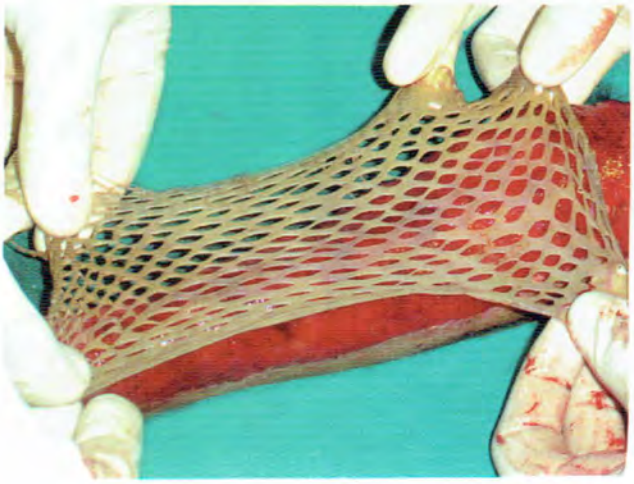


Fig. 14.44C: Meshed graft being applied on raw area

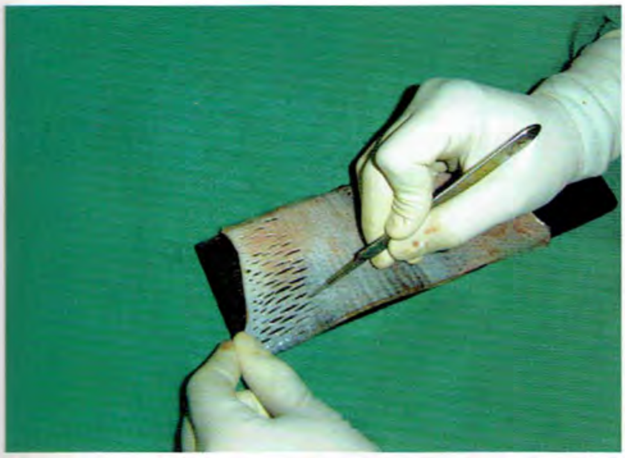


Fig. 14.43A: Meshing of graft being done by hand

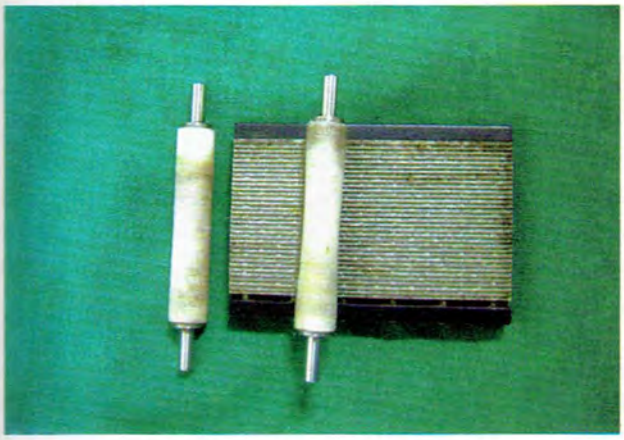


Fig. 14.43B: Padgett's roller skin graft mesher

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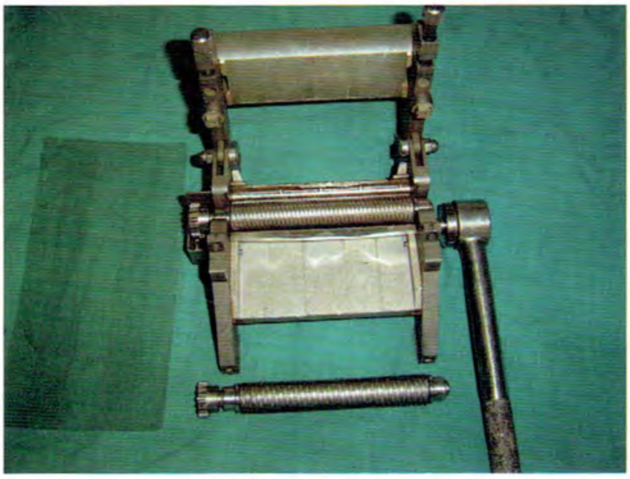


Fig. 14.44A: Zimmer's skin graft mesher

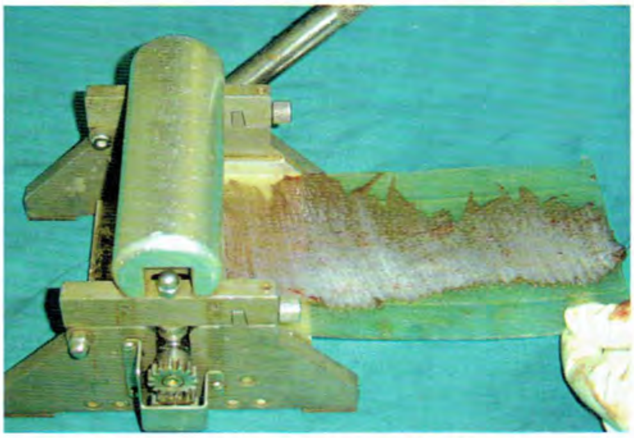


Fig. 14.44B: Graft being meshed by passing it through the mesher

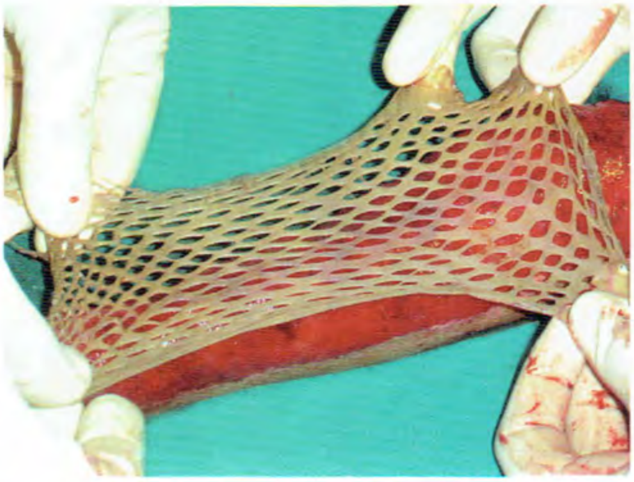


Fig. 14.44C: Meshed graft being applied on raw area

Disadvantages

The disadvantages of meshing are:

1. The checkered board pattern or cobblestone appearance can be displeasing especially in aesthetically important areas like face.
2. The secondary epithelium in the meshed area is not of the same quality as the grafted skin and this is visible for rest of life.

Application of Graft

Before the application of the graft to the wound bed it has to be ensured that proper hemostasis is achieved especially if the unhealthy granulation tissue on the wound bed has been excised with a scalpel. Hemostasis is achieved by using adrenaline saline soaked (1 in 2, 00,000) gauze pieces with pressure dressing or by fibrin glue which is being used of late (Tissel®).[47,48] The graft is then spread over the wound with the help of graft spreader or with the back of forceps ensuring that the edges of the graft are properly spread out as these have a tendency to roll over. This can then be held in place with suture or surgical staples if areas are small or with fibrin glue[47,48] (Fig. 14.45). The graft can also be applied on the raw areas without using the above and covered with nonadherent gauze (Fig. 14.46).

In the past it was a routine to do an occlusive dressing after any split skin grafting because it was considered that pressure over a graft was essential for its 'take'. However, with time and experience with different techniques, it has been clearly demonstrated that a graft can 'take' satisfactorily over granulating areas as long as it is not mechanically displaced or lifted off from its
hematoma or pus formation and that occlusive dr
are not a must. Areas like the anterior chest, abdom
back of trunk where constant motion during resp
may displace the grafts by producing shearing betw
dressing and the wound bed are better treated by ex
method after applying the graft. This is also true
neck (Figs 14.47A to C).

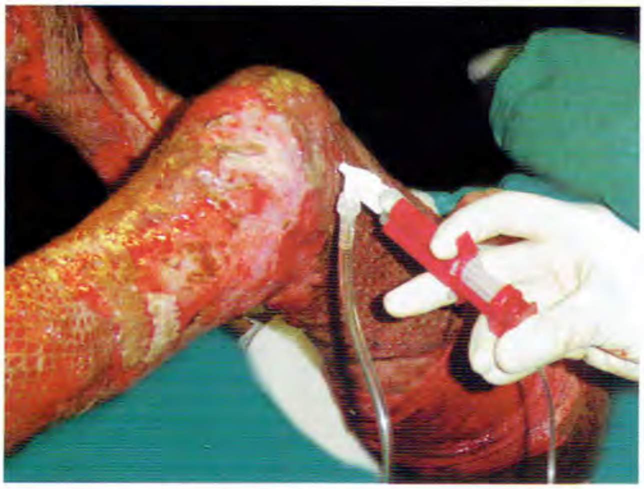


Fig. 14.45: Fibrin glue applied as a spray for anchoring graft to raw area

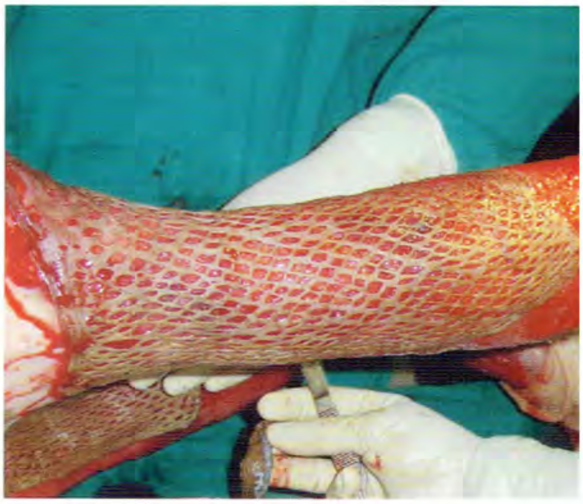


Fig. 14.46: Graft spread on wound as a sheet, edges roll

If dressings are used as in the limbs, it is imper
give an even gentle compression dressing with
immobilization of the extremity (Figs 14.48A and
general principle is to immobilize a joint above and
the grafted area to minimize limb movements and thu
shearing forces. In case of mobile areas like axilla o
stent dressing is advisable.

If the grafts are left without dressing, they sh
inspected daily for initial 4-5 days to check f
displacement or any seroma or hematoma. Howe
meshed graft is used it is advisable to apply a layer of
gauze and a single layer of wet gauze till epithelializ
the interstices has occurred. Otherwise the interveni
areas tend to dry up and epithelial migration is in
The same holds true for stamp grafts.

Skeletal traction can be given to the extremities to
and immobilize the extremity following circumfe
grafting of the limb by exposure technique. Sutu
graft edges is not favored in extensive raw areas as
result in hematoma and at times microabscesses
suture site resulting in graft loss. If adequate graft is av
it is preferred to overlap the adjacent graft edges slig

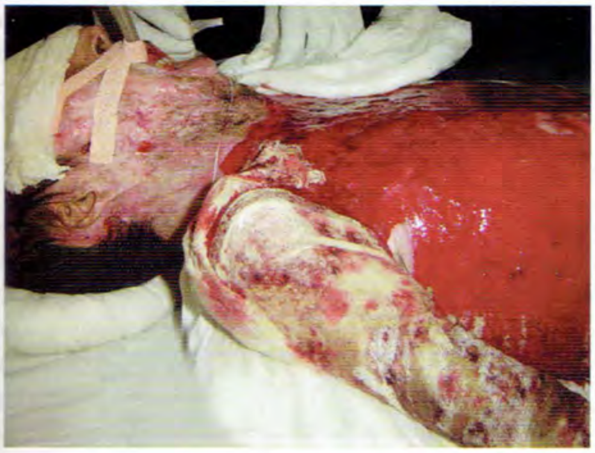


Fig. 14.47A: Granulating wound over chest and neck, ready for grafting

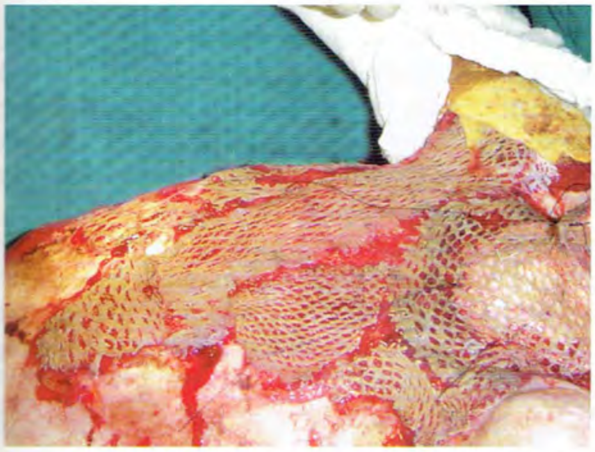


Fig. 14.47B: Graft applied on the raw area over chest and released neck contracture and left exposed

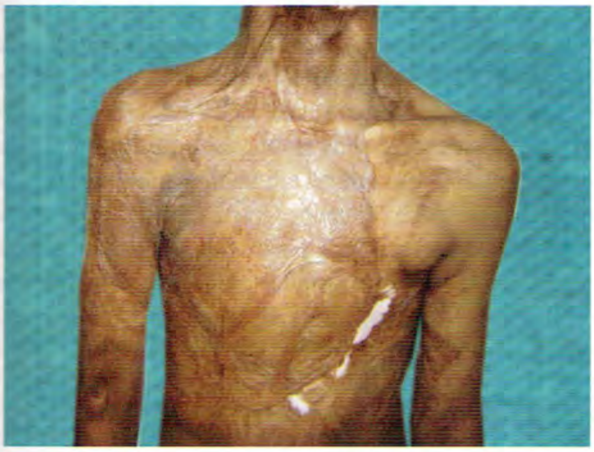


Fig. 14.47C: 2 months postoperative picture of same patient with completely healed areas with no hypertrophy

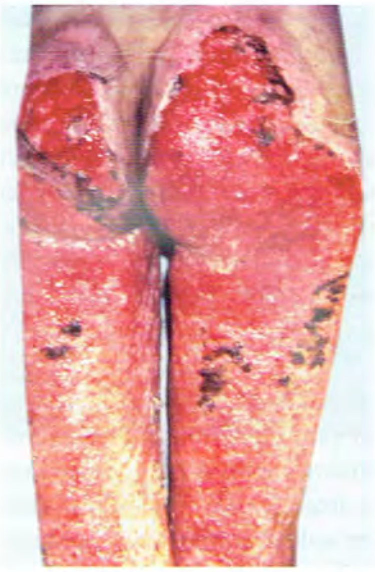


Fig. 14.48A: Extensive granulating wounds over both lower limbs

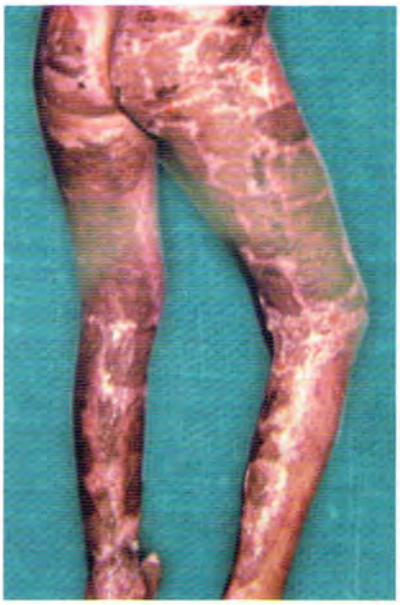


Fig. 14.48B: Wound closure with split thickness skin autografts

as to avoid intervening raw areas. Since the intergraft raw areas heal by epithelial proliferation from the graft margins it results in hypertrophic scarring which is aesthetically ugly and at times functionally poor.

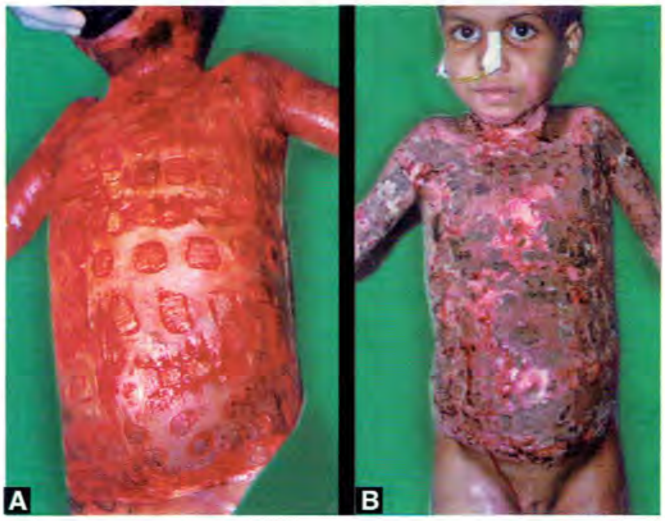
Where homografts are available and donor area is limited, alternate strips of autograft and homograft are applied. In such cases as the homograft starts getting rejected after 14 days, the epithelialization from the margins has already covered quite a distance under the homograft. If homograft is not available and there are many small pieces of graft as in 'postage stamp' grafts, they can all be spread on paraffin gauze with raw surface upwards keeping a distance of 1 cm between two pieces. The paraffin gauze is then reversed and applied to the wound surface so that grafts adhere to the bed. 'Postage stamp' autografts are also useful on multiple small areas of granulation (Fig. 14.49). The Meek grafting is another method of obtaining extensive meshing to produce postage stamp grafts for coverage of extensive areas, as has been described above (Figs 14.50A to D).

Exposure technique is also preferred if the wound cultures show gram-negative organisms as these organisms are known to proliferate in the dark, moist and warm condition provided by a dressing and will float the grafts off the bed because of the volume of pus they produce. If these are kept exposed the activity of these organisms is decreased and chances of graft survival are better.

After the fourth day, usually the time by which the grafts have adhered fairly well to the wound bed, change over to treatment by dressings is advisable.

Delayed Application of Graft

At times in an anesthesized patient it may be difficult to dress certain areas and there may be a risk of grafts getting displaced during recovery from anesthesia as is seen in neck, trunk and face and in heavy patients. In such cases, it is advisable to store the grafts and apply them to the wounds after a day when the patient is fully conscious and cooperative. The grafts can be applied bedside withou anasthesia. Delayed application of graft is preferred if s or granulation has been excised and oozing from th area cannot be controlled.



Figs 14.49A and B: Intermingled skin grafting (Allograft and autograft) leading to complete wound healing

Dressing Change

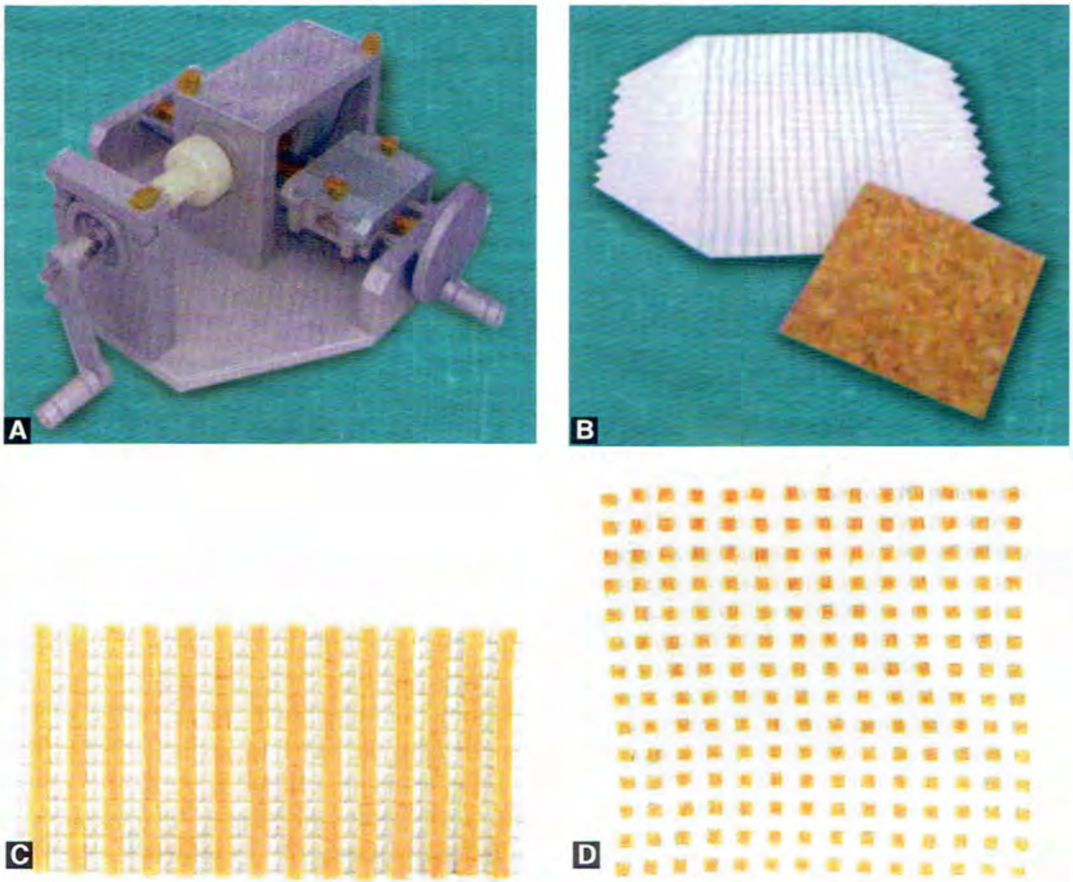
All postburn raw areas are potentially contaminated wo Therefore, it is of utmost importance to change the dressing by 48-72 hours post grafting. This should be with full precautions not to move the grafts. They are dabbed with saline gauze to remove all the exudat express out any seromas through the mesh or by giv slit in the graft. A new dressing of the same type is reap If delayed beyond 72 hours, in the author's experi bacterial autolysis of graft or the purulent collection the graft can lead to loss of significant areas of graf prolong the healing process.

The second dressing change can then be done at 5 post grafting and dried up graft overlapping the norma can then be debrided and exposed dressing can be cha to a closed dressing which will be less bulky. There the dressing can be changed as often as required til areas heal completely. Sutures or staples, if applied, c removed on the third dressing. Immobilization of joint be discontinued after 7 days, once the grafts have take and some movements of joint can then be allowed d dressing change to avoid stiffening of the joints. P physiotherapy with full range of movements shoul resumed only after all dressings are removed and heali complete.

At times for one reason or the other, there can be areas of graft failure. If they are more than 1 cm patch grafting in a second stage may be required. How if they are less than 1 cm wide, even though they m long, trials at repeated grafting may fail. In t circumstances the areas should be cleaned thoroughl dressed with some topical antibiotic over mesh gauze with some amount of topical steroid preparation. Th steroids are known to inhibit epithelialization, they pr the formation of exuberant granulations, over w epithelium anyway can not proliferate. The use of to steroids allows the hypertrophic granulations to settl the epithelium from the surrounding graft grows and sp over the remaining raw areas.

Blood Loss from Donor Areas

It is important to know about the blood loss while harve graft since majority of the burn patients have bord



Figs 14.50A to D: (A) Meek machine for 'Meek grafting' with the cork plate and prefolded gauze (B).The graft is spread on the cork plate and cut into small pieces by passing through the mesher. After the epidermal side of graft is sprayed with an adhesive, the graft is transferred to a prefolded gauze and the cork plate is removed. Expansion is achieved by unfolding the gauze, first in one direction (C) and then in the other direction (D) (*Courtesy:* Humeca Engineering group, Netherland)

hemoglobin levels. It is believed that older patients bleed less compared to younger patients who are well nourished and have a good layer of subcutaneous fat. The back and buttocks bleed more than other areas of the body. Bleeding is more after harvesting thicker grafts than thinner grafts (Figs 14.51A and B). However, on an average, while harvesting split thickness skin graft, there is a loss of 23 ml blood from 5 × 10 cm of donor area, approximately 1% of total body surface area. This was studied by Robinson in 1949. However, it will vary according to the thickness of graft taken.

Graft Survival

Graft survival has been studied in detail during the past century. By definition, skin graft is a piece of skin which is transferred from one place on the body to another after severing its blood supply, lymphatic system and nerve supply. So it is essentially skin transplantation and it derives its blood supply and innervations from the recipient area.

Serum Imbibition

Immediately after a skin graft is applied to the wound bed, the process of vascularization of the graft starts. Initially, the graft depends on the serous exudates or plasma which leaks out from the recipient venules, arterioles and capillaries and fills up the space between the graft and host bed. This plasma is absorbed by the graft and is the source of its nourishment. This process was called 'phase of serum imbibition' by Converse, Uhlschmid and Ballantyne[49] and originally called 'Plasmatic circulation' of graft by Alubscher and Goldman.

Once the graft is lifted off the donor area, its vessels go into spasm squeezing out old blood and serum. When it is applied on the host bed, the cut ends of these vessels and

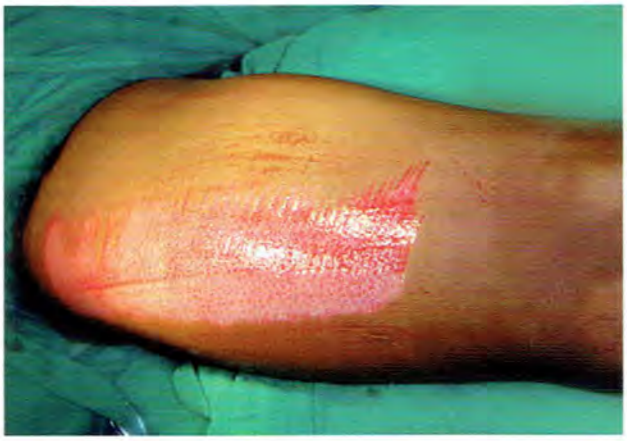


Fig. 14.51A: Donor area of thin SSG—Bleeding points are small but numerous

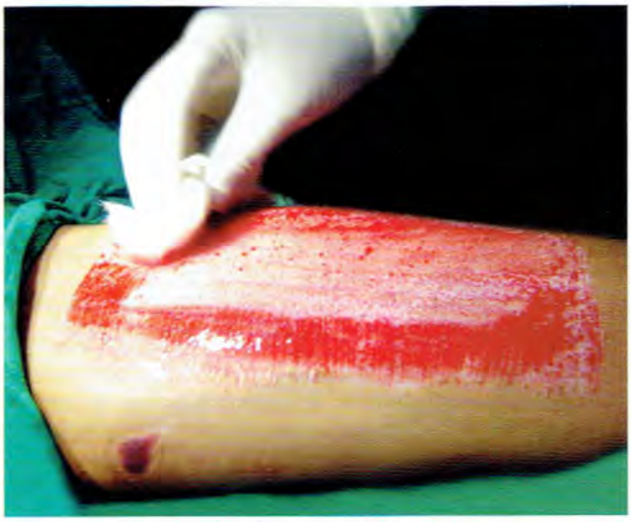


Fig. 14.51B: Donor area of thick SSG—Bleeding points are larger but fewer

the porous dermis starts absorbing the serum collected between the graft and host bed passively. This makes the graft edematous and its weight increases with anaerobic metabolism which accumulates the waste products in the graft till 48 hours until the venous drainage starts. In this phase, there is growth of endothelial cells from the host into the graft as well.

Graft Revascularization

There have been various theories regarding how the graft gets revascularized. The earliest was the theory of 'Abouchment' given by Beat (quoted in[50]) in which he noted microscopic anastomosis between host bed and graft. This was supported by Thiersch (quoted in[26]) who termed the process of anastomosis between cut ends of host v and vessels of the graft as 'inosculation' in a full thic graft.

Twenty five years later, many researchers (quote proposed that the original vasculature in the skin degenerates and endothelial cells and capillary buds the host invade the graft by 9 hours and form a va network and flow through the graft is established b 4th postoperative day. These were confirmed by va histochemical, injection and histologic studies.

Another theory proposed by Henry [51] stated that th the original vasculature of the graft degenerates, the ac basal lamina persists and acts as a condui endothelialization to start from the host capillary bud the skin graft. This is also seen within 48 hours.

Thus, all the theories are by and large proposin same explanation for the graft revascularizatio summarize, the vascular connections establish with hours after grafting allowing inflammatory cells to i the graft. By 4th postoperative day blood inflow th the graft from the host bed is established. The graf appears pink and blanches on pressure. This flow con to increase till venous outflow from the graft back host bed is also started and by 1 month after graftin vascularization phase is completed.

The revascularization process is similar in both thickness and full thickness skin graft. However, thickness grafts survive longer without revasculari than a full thickness graft because they contain fewer ce elements to be nourished. The serum imbibition has t place through a shorter distance through the dermis is thinner. In a full thickness graft, on the other han full thickness of the dermis acts as a barrier to the diffusion and hence, it depends more on the supply th the edges of the host bed with which it is contact. Th all granulating raw areas or areas with poor vascula thin split thickness graft survives better than a thicke and should be used.

Once the process of revascularization is underst is easier to understand why a hematoma or seroma u graft inhibits its 'take' though they allow initial imbibition. They prevent the vascular ingrowth fro host bed into the graft which eventually dies out. Thu important to evacuate any hematoma or collection the graft before 72 hours by inspecting it on the first dre which is advisable by the 3rd day. After evacuatin hematoma, the undersurface of the graft in that area it is lifted off the bed, also needs to be scraped or r with a gauze so that the tenuous lining of the proliferating epithelium which forms on the undersurface of the graft over the hematoma is removed. Otherwise it can inhibit the graft 'take' once it is kept back on the host bed.

Graft Maturation

After the graft is vascularized it matures over a period of time. An immature graft is soft, pink in color and blanches on pressure and may be thick and shows scaling. The process of maturation takes about 6-12 months. During this phase of maturation both the wound and the graft appear to undergo contraction. This process of graft contraction is actually a manifestation of the wound bed contraction mediated by the myofibroblasts. The myofibroblasts are differentiated fibroblasts with features resembling the smooth muscle cells, hence they contract the bed. This, in turn, causes the graft to contract to the dimensions of the underlying bed. This process is called 'Secondary Contraction' of a skin graft.

Primary contraction of graft, on the other hand, is an inherent property of every skin graft and occurs immediately after the graft has been harvested. It is due to recoil of the elastic fibers in the dermis. Therefore, primary contraction is more in a full thickness graft as compared to a split thickness skin graft because of greater amount of dermis and greater amount of elastic fibers.

A full thickness graft has minimal secondary contraction compared to a split thickness graft. The reason for this appears to be the full thickness of dermis in these grafts (FTG) which inhibits myofibroblasts differentiation. In a split thickness graft, the lesser the amount of dermis, the lesser is the inhibition of myofibroblasts differentiation, therefore the bed tends to contract more. Thus, after application of SSG to post burn raw area, splintage and pressure is required for a long time postoperatively to avoid the secondary contraction. Besides greater secondary contraction in SSG, it tends to have lesser growth potential. This is important in the pediatric age group. In children with burns, treated with SSG, contracture formation is more as the graft does not grow with age and with respect to the rest of the body. An FTG on the other hand has a better ability to grow with age but is rarely used in pediatric burn wounds for reasons already stated.

In darker races, it has been observed that the graft is susceptible to hyperpigmentation. Thus, exposure to sun should be avoided for at least 1 year after grafting.

Reinnervation of Graft

The nerve fibers in the graft degenerate in 3 days after transfer to the recipient site. The new nerve fibers invade the graft from the periphery as well as the recipient bed at 40 days travelling along the empty Schwann sheaths or along the blood vessels. At 2-3 months as these fibrils increase in number they reach the end organs in the graft, i.e. hair follicles, sweat glands and sensory end organs and gradually the sensation returns in the graft. Pain is the first sensation to return followed by touch, temperature and tactile discrimination, although the sensation does not match that of the normal skin. In fact, in the first year of post grafting, the graft may be hypersensitive. The sensations are better in full thickness (FTG) as compared to a split thickness (SSG) skin graft.

Sweat glands present in the dermis of the skin graft start functioning only after the reinnervation of the graft is established. Therefore, there is no sweating in the graft for 2-3 months post grafting. Till such time, the graft remains dry and scaly and needs to be protected by moisturizing creams. Once the sweat glands are reinnervated, their behavior is similar to that of the recipient bed and not the donor site.

Hair growth is rarely seen in thin SSG because of damage to majority of the hair follicles. But in a thick SSG and FTG, hair growth resumes and resembles that of the donor area. This is significant when FTG from scalp is used for eyebrow reconstruction following burns.

Complications of Skin Grafting

1. The main complication of skin grafting is the failure of graft to 'take'. The various causes for graft failure are:
   a. Infection: Infection is the most common reason for graft failure especially with beta-hemolytic *Streptococcus*. Therefore, it is imperative to have a wound culture-sensitivity report prior to grafting.
   b. Hematoma: Hematoma under the graft prevents the graft from getting vascularized. Therefore, proper hemostasis in the bed prior to application of graft should be ensured. In case it cannot be achieved, graft can be stored and applied after 1 or 2 days when the bleeding stops.
   c. Ineffective immobilization: Failure to immobilize the grafted area is another important cause for failure of graft 'take'. Therefore, it is important to avoid shearing between the graft and the wound bed and for this proper measures as discussed above have to be taken.

d. Poorly vascularized bed: It is not able to sustain a graft and leads to graft failure.

2. Blister formation and breakdown in graft occurs in the extremities especially weight bearing lower limbs in which the increased hydrostatic pressure causes venous congestion in graft. This is also seen as purple discoloration of the graft.
3. Itching is a persistent problem of grafted patient. It is due to drying of the graft.
4. Repeated scaling in the graft is seen because of absence/ diminished number of sweat and sebaceous glands in the graft. Hence, they require lubrication for rest of the life.
5. Contractures result from failure to splint and apply pressure over the graft which allows secondary contraction of the bed and graft. This process lasts for 1-2 years.

Postoperative Care of Graft

Once the wounds have completely healed, the grafts have to be kept lubricated with some oil massage and require a continuous pressure using pressure garments which prevent inter-graft hypertrophy. In lower limbs, the pressure support is essential for 3 to 6 months. This can be given by crepe bandage or pressure garments. Apart from preventing hypertrophy in grafts, they also prevent venous congestion and breakdown in the graft. They have to be worn throughout the day and night especially during ambulation.

At 10 days postoperatively, hanging of feet over the side of bed can be allowed for short intervals of time followed by elevation. Weight bearing in grafted lower limbs can be started only after 2 weeks and gradually by 3 weeks walking can be resumed but only with protection of a pressure garment or crepe bandage.

The grafted area can be left exposed if there is no loss by 10 days. By 14 days patient can be allowed to have a bath. By 3 weeks the graft is usually strong enough to withstand reasonable wear and tear.

Ideally, after the skin grafts are harvested they are immediately applied to the raw areas. In case the wound has been excised or debrided and hemostasis is not achieved properly, it is a waste to apply the graft on a bleeding bed. In these circumstances, the graft can be stored for a day or two and applied to the raw area later in the ward. Sometimes, an excess of graft may have been harvested and this excess graft is also stored till such time that it may be used if there is loss of some previously applied graft.

The most common and practical method of storing autograft is in the refrigerator at 4°C. Chilling is known retard rate of metabolism and prolong survival. The gra are spread on paraffin gauze with raw surface upwa and the paraffin gauze is then folded on itself and then furt wrapped in saline gauze. This is then placed in a ster bottle which is kept in the refrigerator at 4°C. Grafts can stored up to 3 weeks in this manner and thereafter discard With increasing time interval, the tissue viability decreas Care should be taken not to freeze the graft as freezing temperatures below 0°C causes mechanical rupture of ce and disruption of cytoplasmic contents. The reader is advis to see the details of skin banking elsewhere in this book

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Chapter
15

Surgical Excision o Burn Wound and Skin Graftin

Pramod Kum

INTRODUCTION AND HISTORY

Ever since fire has been known to human beings, not only it has been an undoubted boon but also a bane to some extent. The treatment of oldest (59,000–48,000 BC) known historic burn wounds with herbs and roots has been recorded in cave paintings of Neanderthal man. Later in 1500 BC, Egyptian Smith papyrus used salve of resin and honey for treating burn wounds.[1] Sushruta, the famous surgeon of ancient India (800–600 BC) and regarded all over the world as father of plastic surgery, in his book Sushruta Samhita[2] (Section 1: *Sutrasthana*, Chapter 12: *Agnikarma Vidhi Adhyaya*) has described the procedure of branding by fire (*Agnikarma vidhi*), burns (*Dagdha*)—types and treatment, suffocation due to smoke (*Dumopahata lakshna*) and sun burn (Atapadi dagdhana).[2]

Although Jackson (1954) is regarded as the pioneer of early burn wound excision and grafting, Janzekovic[1] (1970) showed that early return to work (about 1 week post burn) is possible after tangential excision and split skin grafting by day 3 to 5. In 1970, a brief article in Journal of Trauma by Yugoslavian plastic surgeon, Zora Janzekovic, went unnoticed. In 1974, Monafo advocated excision and grafting for larger burns. In 1974, Burke used fascial excision for large burns with a huge increase in survival and decrease in hospital length of stay.[1] Later, Janzekovic gained considerably more attention when she reviewed her experience again as the Evans lecturer for the American Burn Association in 1975.[3] Until her observations were published, nearly all excision and grafting procedures were limited to full thickness burns and skin and subcutaneous tissues were removed beneath the burn to the level of the investing fas Due to disappointing results with excessive graft l following infection from adjacent non-excised area, she al with many others abandoned early operation. Dr Janzeko knew that deep donor site could be over grafted with thinner autograft, thereby hastening the healing a improving the appearance. Realizing that graft would surv on viable dermis, she applied this technique to dermal bu by progressive shaving off of thin layers of burnt der until she reached a bed that appeared viable and immediately autografted it successfully. Between the ye 1961 and 1974, Dr Janzekovic treated 2615 of 4370 bur patients with this form of early tangential (or sequent excision and sheet autografting. Most of the burns w small (less than 20% TBSA) but the hospital stay, pain need for reconstructive surgery reduced considerably lead to her opinion "Aesthetic disability" reduc considerably.[4] Though Tanner (1964) developed mes skin grafting technique, Alexander (1981) showed advanta of using widely meshed autograft and its coverage w cadaveric graft. Development of artificial skin, Integra (bi-layered skin substitute made from collagen with pla cover) by Burke (1981) and development of tissue cultu grown skin by Bell made a substantial improvement in management of larger burns using excisional surgery.[1]

Davies in 1990 reported that in India 22% of bu patients died in the first 48 hours due to inappropriate inadequate fluid therapy, 36% by day 7 post burn due circulatory or respiratory complications and virtually all deaths during weeks 2, 3 and 4 after burns were due overwhelming sepsis.[5] Incidence of contractures and hypertrophic scar is more if skin grafting is not done early and wounds are allowed to granulate. Baxter[6] documented the possibility of minimizing wound infection by topical use of 1% silver-sulfadiazine. Barret and Handerson[7] (2003) in their study concluded that burn wound excision significantly reduces bacterial colonization. Patients who underwent topical treatment and delayed burn wound excision exhibited greater bacterial colonization and increased rates of infection. Burke et al[8] noted that time required for closure of the open burn wounds using primary excision and skin grafting was reduced to $1^{1}/_{2}$ time compared to topical therapy alone.

Modern advances and better understanding of burns treatment, especially in burns resuscitation, infection control and nutrition along with early excisional surgery and skin grafting have led to significantly reduced overall morbidity and to some extent burn wound sepsis and mortality.[4] Uninfluenced by the survival statistics, excisional surgery has become the treatment of choice for all deep dermal and full thickness burns due to reduced cost of treatment, better functional result, early rehabilitation with return to work and a reduction in long term morbidity.

TYPES OF BURN WOUND EXCISION

The burn wound excision may be broadly classified into:

1. *Primary excision*: It is done within 3-7 days following burns. It is actually a delayed primary excision because it is done after the third day of burns for extensive burns. This is further sub-classified into Tangential, Sequential and Fascial excision, depending upon the technique used.
2. *Secondary excision:* This is basically a procedure for preparing the wound bed for grafting by removing the eschar. It is synonymous with surgical toilet or debridement.

TYPES OF PRIMARY EXCISION

1. **Tangential excision**: It is used typically for deep dermal burns. In this technique, the wound is excised with Watson's skin grafting knife till healthy viable dermis is reached over which split skin graft is applied immediately.
2. **Sequential excision**: It is also used for deep burns in which Watson's skin grafting knife is used to sequentially excise all necrotic tissue until viable tissue is encountered which can be either viable deep dermis or viable fat or fascia. This area is also immediately skin grafted. This technique is most effective for mixed deep dermal and full thickness burns in which sequential excision helps in saving the viable dermis or fat which would otherwise be sacrificed if the entire burned area were to be removed with a surgical blade.
3. **Suprafascial full thickness excision**: It is indicated for full thickness burn wounds. In this, full thickness of burned skin with subcutaneous tissues is excised with a surgical knife till the deep fascia. This can be immediately grafted because fascia is a healthy bed for graft 'take'.
4. **Subfascial excision:** It involves excising till deep to the deep fascia, thus reaching the muscles. In these cases, the chances of graft 'take' are poorer compared to suprafascial excision.
5. **Escharectomy and delayed skin grafting**: This was practiced earlier for partial and full thickness burns in which the nonviable eschar is excised with a skin grafting knife till minimal bleeding bed is encountered. This bed is, then, covered with a biological dressing and reassessed after 48-72 hours to look for remaining dead tissue which is again excised and dressed till healthy granulation tissue appears. This is secondarily grafted. This procedure is somewhere between primary excision and secondary grafting following conservative management of burn wounds as it takes 7-10 days for the healthy granulation tissue to appear following excision of dead tissue. This is, however, not practiced anymore. It is suitable for cases where primary excision cannot be done due to medical reasons or associated injuries.

ANATOMICAL AND PHYSIOLOGICAL BASIS FOR EXCISION

The principle and type of excision can be explained by the anatomy of skin and depth of burn. For excisional purposes, the depth of burns is broadly classified into partial thickness and full thickness burns. The partial thickness burns are those which involve the epidermis and upper half of dermis and they heal without intervention in 10-14 days. Full thickness burns are those which involve the entire epidermis and dermis and these do not heal on their own and require surgical intervention. In between these two are deep dermal burns which involve the epidermis and two thirds of dermis and these heal in a long time with severe hypertrophic scarring and contracture formation. So in these cases if tangential excision is done it would not only improve morbidity but even mortality is reduced dramatically. The deep dermis is still viable, therefore it needs to be covered with split skin graft following excision.

INDICATIONS

Early excision is especially appropriate for flame and dry heat injuries, whereas a more conservative approach is needed for scald injuries which are often associated with less severe second degree burns that are likely to heal well without excessive scarring. The results of a prospective randomized study in a pediatric population showed that scalded patients should be observed for approximately 14 days, after which the need for operative intervention can be reassessed.[9-13]

Size of burn: It is a general agreement that small full thickness, indeterminable and deep dermal burns can be excised safely and grafted with good result (short hospital stay, reduced morbidity, reduced burn wound pain and sepsis, reduced nurses turn over in critical care area and reduced scar related problems). Larger burns due to paucity of skin donor area may require homograft, synthetic skin substitutes or cultured epithelial autograft. In massive burns (>60%TBSA) the problem of inadequate donor area can be tackled by using widely expanded (meshed 1:4) autologous split-thickness graft that can be overlaid with unexpanded (with hand meshed) homologous (cadaver) split skin graft.[10,11,14,15]

Burns of 30-40% BSA may be excised and covered with autograft in two sessions at 10-14 days interval. The excision of 15-20% BSA in one session is safe in healthy individuals, although some surgeons set the limit even up to 40%.[11] In life threatening burns, one should aim at covering larger areas (i.e. area easy to excise and easy to graft e.g. trunk) rather than getting better functional result, (i.e. excision of hand burn and finger burn that requires more time to cover less area).[16]

Timing of excision relative to changes in the wound itself is crucial to minimize risks: Timing for primary excision: Tangential excision is usually done between 3rd and 5th day following burns. The rationale for not doing it earlier is to give adequate time for resuscitation especially in extensive burns and to avoid unnecessary excision of deeper tissues where perfusion is restored after resuscitation is completed. Excision is not done after 5 days because there is a significant bacterial colonization of wound by then which may inhibit graft 'take'. After 7 days, there is softening of the surface of wound which makes excision technically difficult.

Colonization of the wound develops during the first we and manipulation of the colonized or infected wound ru an increasing risk of bacteremia. The reduced rates of wou infection and sepsis in past three decades have been t result of many factors, of which early excision has be advocated as one of the major factors. Barret and Handersc (2003) have shown that burn wound excision significan reduces bacterial colonization. Patients undergoing topi treatment and delayed burn wound excision exhibit grea bacterial colonization and increased rates of infection. Th have also advised acute burn wound excision for all f thickness burns. Desai[18] et al (1990) have advocated t near-total excision of large burn within first 24 hours reduc blood requirement and morbidity without adversely alteri hemodynamic stability and increasing mortality risks. Th have shown that blood loss in large burn injuries (m than 30% TBSA) was significantly decreased when surgi excision was performed within the first 24 hours after inju compared to those performed between the second a sixteenth day after injury. Reduced blood loss was notic in second 24 hours after injury; however, this was n significant compared to blood loss between post burn d 2 and 16. Blood loss in burns treated conservatively w grafting on granulation tissue was also less when compar to blood loss from excision during post burn day 2 throu 16. Hence, the size of the planned excision must determined based on a clear understanding of increasi risks of blood loss and bacteremia.

Early excision and grafting are not justifiable in conditio like charred hand where paratenon is affected and fl surgery is indicated. Excision and grafting should be avoid in the presence of inhalation injury.

Criteria for Early Excision

The diagnosis of deep burn must be established (only are that do not blanch on applying pressure by finger tips excised), patient should have enough donor area a patient's condition should permit major operation. Inhalati injury is often considered as contraindication for ea excision and grafting.

GENERAL PRINCIPLES FOR BURN WOUND EXCISION AND GRAFTING

- The patient must be hemodynamically stable bef considering excision.

- The potential for significant blood loss must be recognized and adequate amount of blood and its components, if indicated, must be available before starting excision. Significant blood loss in a major burn should be replaced with blood products.
- Pulmonary problems require a plan of management in the operating room. In post-operative period, mechanical ventilation may be required.
- Hypothermia must be prevented during surgery. All the areas should not be exposed at a time. Operating room temperature should be maintained between 80 and 85°F and fluid warmer should be used. Patient's body temperature should be monitored.
- The stress induced by anesthesia and surgery must be limited to that which the patient can safely tolerate. Each operative period should be limited to 2-3 hours.

As suggested by various authors[4,19,20] the following points should be considered when deciding to proceed with operation:

1. Experienced surgeon is required for early excision. Inadequate excision can lead to loss of graft placed over it and add to the size of donor area thus created to the extent of burn and may necessitate another operation.
2. Small burns that eventually heal and offer little threat to life if allowed to heal over several weeks: In this group patient selection should be such that if excised, operative mortality remains non-existent.
3. Small burns that can be managed and taken care of by the patient till healing at minimal cost without missing work, may be treated conservatively.
4. Small deep burns can be treated on an out-patient basis initially and then, excised electively and grafted on a day surgery schedule.
5. Loss of income due to hand and feet burns can be significantly minimized by early excision and skin grafting.
6. Life threatening associated problems including trauma must be well controlled so that operation can be done with low morbidity and essentially no mortality. Herndon et al[20] have shown that the presence of pre-admission shock and inhalation injury were early determinants of mortality with secondary renal, pulmonary or cardiovascular collapse being the later predictors of mortality.
7. Large superficial burns with scattered small deep components are best treated conservatively until the shallow areas are healed.
8. *To avoid problems of excessive wound related pain and slough,* excision and grafting may be one of the options. Thompson et al,[19] based on their study of effect of early excision on major thermal injury, have indicated that results with early radical excision and grafting of major burn wounds (>50%) may prove to be equal to or better than those achieved with conservative methods of topical antimicrobial, delayed eschar separation and grafting.
9. *Burns over sole and palm*: Though early excision and grafting was recommended as the preferred form of treatment in patients with deep second degree and third degree flame burns of dorsal hand[21], it is generally agreed that tangential excision and spilt skin graft is not advised on palm and sole as there is a high possibility of damage to the digital nerves and vessels. Singh and Prasanna[22] in a study of five children with burns of feet have found that the tangential excision and split skin grafting for acute burns of feet helps in avoiding pain associated with repeated dressing, reducing the need for reconstructive procedures and hospital stay. It also allows preserving sensate unburned skin and normal growth of the foot.
10. *Burns over scalp:* Tangential excision of the scalp is not practiced widely. Dickson et al[23] in their study concluded that it is preferable to manage scalp burns conservatively and grafting as a secondary procedure. The earlier reports of tangential excision of scalp and grafting showing that it promotes faster healing, reduces chances of hypertrophic scar and preserves hair growth have not been confirmed by the study of Dickson et al.[23] Furthermore, in their study, the major disadvantage of this technique had been recurrent blistering, breakdown and infection. True milia cyst formation was not encountered in their study.
11. *Burns of face:* Wound excision and grafting is indicated for face burns that do not heal within 3 weeks and that the procedure yields results that permit the burn victims to return to society and minimizes the time off work or out of school.[24]

INVESTIGATIONS

1. Hematological: Hemoglobin, total and differential leucocytes count, platelet counts, Preoperative coagulation profile (if indicated)
2. Routine urine examination (sugar, protein and microscopic examination)
3. Bacterial culture: Wound swab, and if indicated blood culture

4. Serum electrolytes
5. Serum protein level (albumin, globulin), Immunoglobulin level (IgG, IgM)
6. Renal function tests
7. Myoglobin in urine (if urine is smoky or tea-like coloration), Investigation for rhabdomyolysis: creatine kinase (CK), aldolase, lactate dehydrogenase (LDH), serum glutamic-pyruvic transaminase (SGPT), and other enzymes.
8. In electric burns: ECG, creatine kinase (CK; also known as phosphocreatine kinase or creatine phosphokinase-CPK), and CK-MB.
9. Arterial blood gas analysis, X-ray chest, Bronchoscopy (if indicated)
10. Investigations to rule out other organ dysfunction (if indicated)
11. Investigations for co-morbid condition and pre-existing illnesses.

TANGENTIAL EXCISION

Tangential excision (Figs 15.1A to 15.5D) is indicated for deep dermal wounds. In tangential excision burnt dermis is excised with modified Humby's skin grafting knife (e.g., Watson's)/hand held dermatome or power driven dermatome. Since excision is done tangent to the wound and often multiple thin slices of the tissue are excised till viable bleeding tissue is reached, the method of excision is called tangential excision.

Anesthesia during excision and grafting: During excision following anesthesia related problems are encountered:

- Setting up of intravenous line: Due to burn wounds, it is difficult to get IV line at usual sites and to fix it by adhesive tape. Very often jugular line is started during anesthesia. Central line is started as the last option. Protecting the line during surgery and changing patient's position under anesthesia may be difficult.
- Patient monitoring: Continuous monitoring of ECG, pulse and blood pressure may be difficult in burns patients as compared to other patients. This may be due to the pattern of burn, extensive nature of the burn and unusual position of the patient on the operation table. By using a precordial stethoscope or use of esophageal stethoscope one can continuously monitor heart beat. Continuous monitoring of blood pressure may be done by starting an arterial line as the last option. It may be difficult to

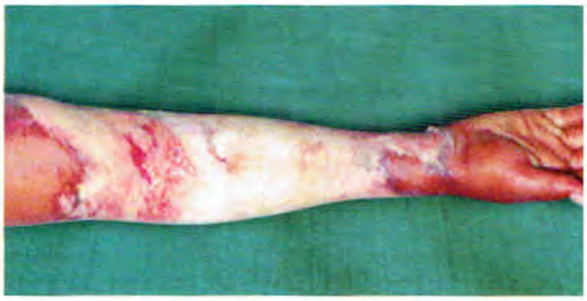

Fig. 15.1A: Preoperative view: Deep dermal burn of right
extremity—Extensor aspect

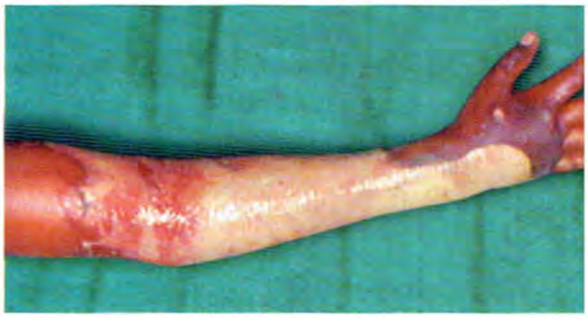

Fig. 15.1B: Preoperative view: Deep dermal burn of right
extremity—Flexor aspect

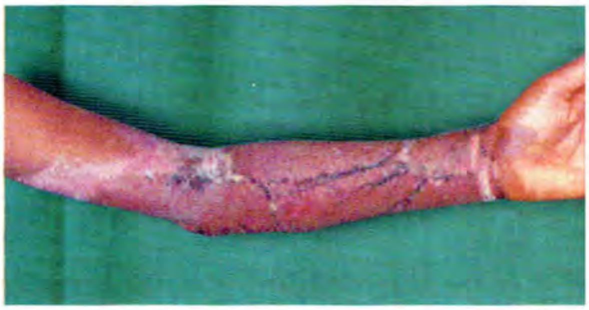

Fig. 15.1C: Postoperative view of same patient following ta
excision and grafting of deep dermal burn of right upper extr
Extensor aspect

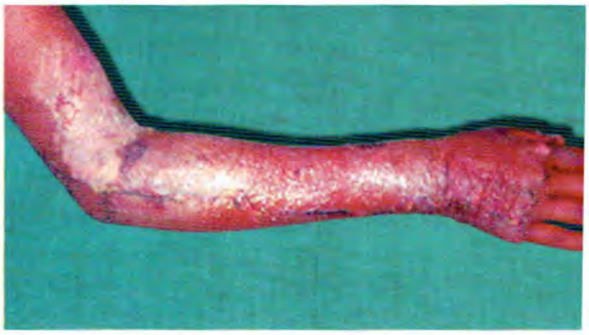

Fig. 15.1D: Postoperative view—Flexor aspect

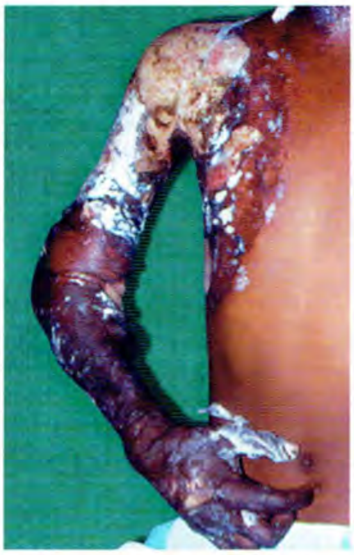

Fig. 15.2A: Deep second degree burn of right upper extremity

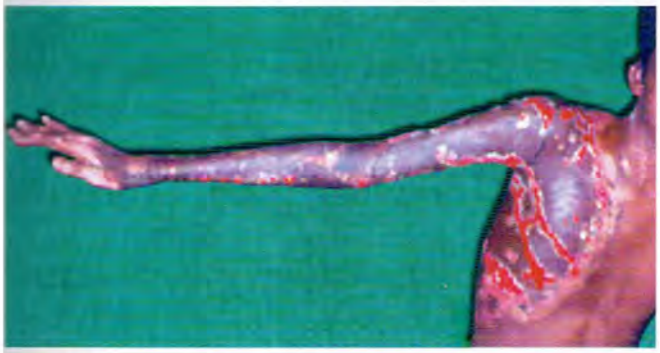

Fig. 15.2B: Result after tangential excision and split skin grafting

place standard ECG electrodes. Therefore, ECG leads are placed at a position that is convenient to both the anesthesiologist and the burn surgeon. Many a times, the site of the sphygmomanometer cuff and finger probe for monitoring oxygen saturation has to be changed from one place to other to enable surgeon to complete the operation.

- Difficult intubation: Intubation may be difficult due to upper airway edema. Once intubated, fixation by adhesive tape may not be possible in facial burns. In this situation the endotracheal tube is tied around the neck with a bandage and the bandage is fixed on the tube at an appropriate site with an adhesive tape.
- Muscle relaxant: In acute burns (like immobilized patients, or those patients who had repeated sepsis) the use of Suxamethonium (succinylcholine) is known to be potentially dangerous due to exaggerated hyperkalemia.
- Sudden blood loss from multiple sites during excision may produce serious problems during anesthesia. Excision may have to be stopped for some time if deemed necessary by anesthesiologist. Large quantities of fluid may need to be transfused in a short duration.
- Hypothermia: Uncovered burnt areas, excised areas and donor areas result in quick loss of heat. Keeping the areas covered that are not being operated, using fluid warmer for fluids being transfused, monitoring patients temperature, controlling operating room temperature, using warm saline mops and limiting the duration of surgery reduces the problem of hypothermia.
- Hypovolemia: Sudden loss of large volumes during surgery may cause oliguria, anuria and finally renal failure. Urinary output measurement at the beginning of surgery, hourly during the surgery and in postoperative period helps in minimizing the problems.
- Postoperative ventilatory support: Maintenance of oxygen saturation may be difficult due to inadequate chest wall movement or chest complications making it necessary to ventilate these patients in postoperative period.

Technique of tangential excision: First of all, the area of deep dermal burn to be excised is mapped and marked (Figs 15.3A, 15.4A and 15.5A). For tangential excision with Watson's modification of Humby's skin grafting knife, the knife is set to take thick split skin graft. Saline is used to keep the area wet and help in easy movement of knife. Slices of burned tissue are excised till viable tissue with brisk bleeding is encountered (Figs 15.3B and C, 15.4B and 15.5B). Viable dermis appears shiny white in color with multiple bleeding points. Small deeper (3rd degree) burnt areas in the deep dermal burn wound may be excised tangentially till viable bright yellow fat is exposed and nonviable brown fat is completely excised. 'Take' of skin graft over the fat is relatively poorer as compared to that on viable dermis. To avoid sudden loss of a large volume of blood, one smaller area is excised and perfect hemostasis is achieved before starting excision over another area. Topical application of adrenaline solution for 5-10 minutes and then use of slow water jet over excised area to localize bleeding points for bipolar cauterization are commonly used to achieve hemostasis. Exposed viable dermis and other tissues are covered immediately by adrenaline solution soaked sponges to avoid its desiccation followed by cover with split thickness skin graft (Figs 15. 3E and F, 15.4C and 15.5C).

Smaller areas of burn surrounded by unburnt skin are difficult to excise by large size knife. Hence, smaller knives like Silver or Goulian-type Weck knife is used. The author

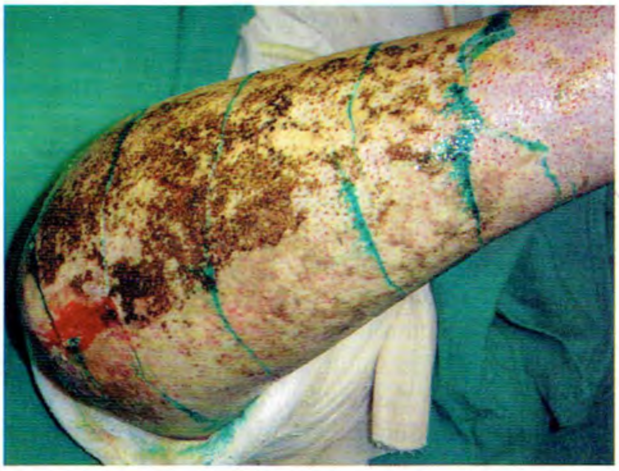

Fig. 15.3A: Areas of deep dermal burns mapped in forearm prior to excision

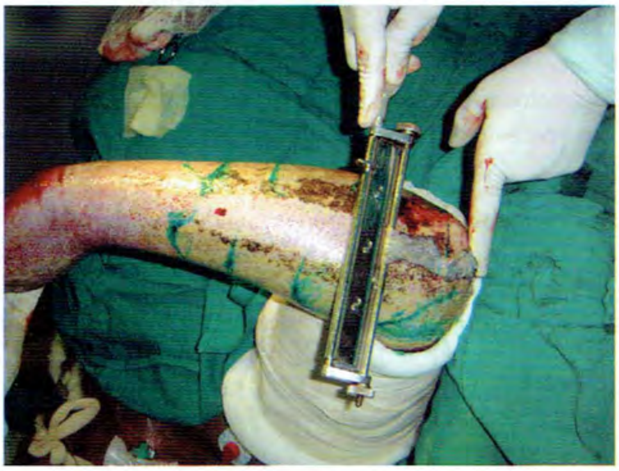

Fig. 15.3B: Watson's knife used to excise dead skin layers only in the mapped out areas

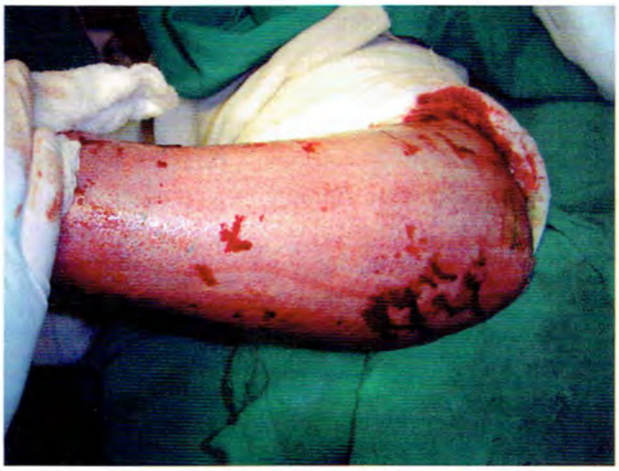

Fig. 15.3C: Healthy reticular dermis layer reached

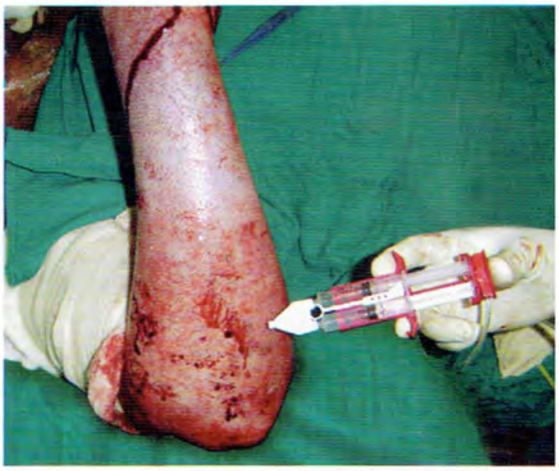

Fig. 15.3D: Fibrin sealant applied locally for hemost

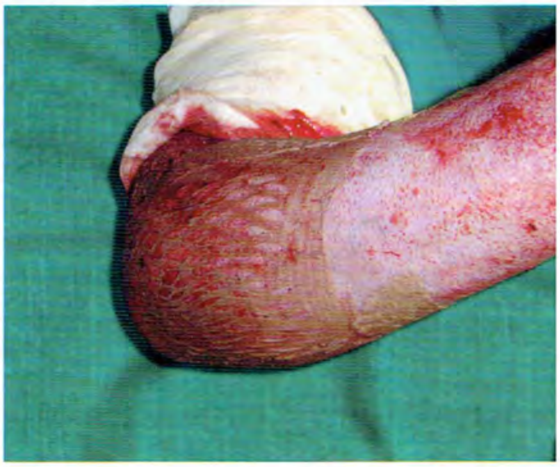

Fig. 15.3E: Split skin graft applied after bleeding is cor

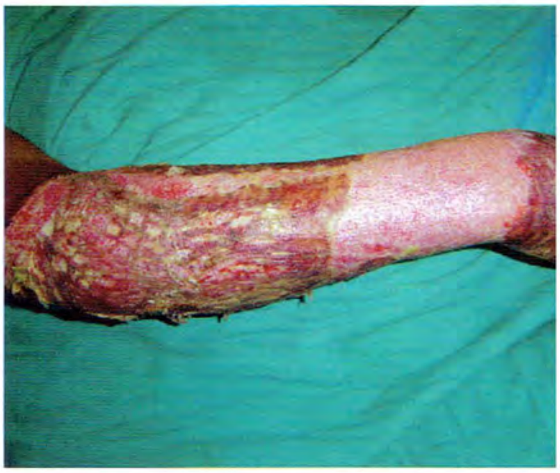

Fig. 15.3F: Second postoperative dressing after 8 days graft fully taken up and area healed

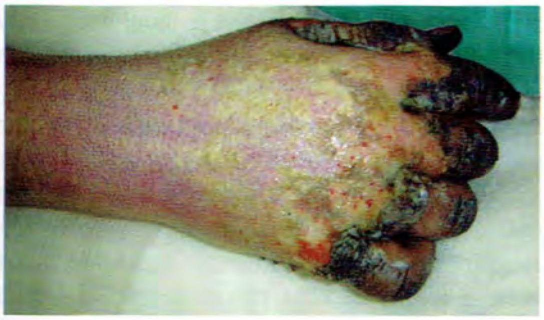

Fig. 15.4A: Deep dermal burns of dorsum of hand

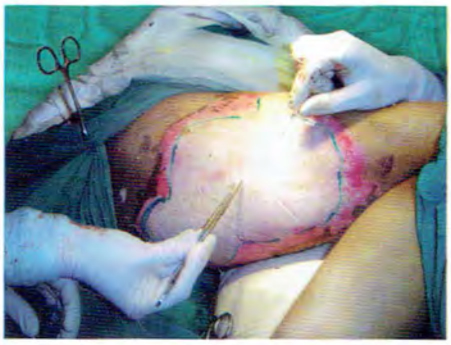

Fig. 15.5A: Deep dermal burn over thigh – Area marked

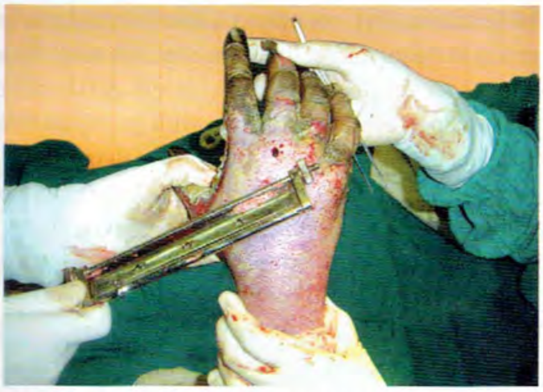

Fig. 15.4B: Tangential excision being done with Watson's knife

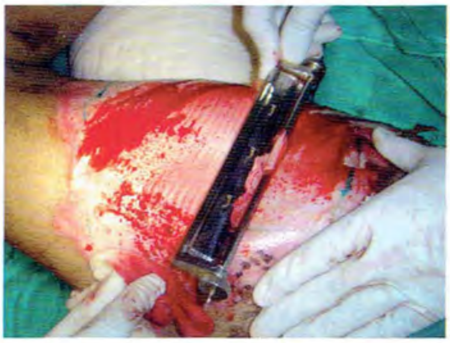

Fig. 15.5B: Dead layer tangentially excised

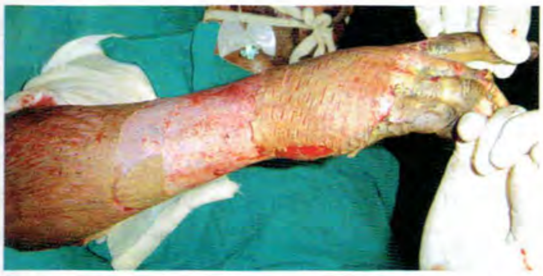

Fig. 15.4C: Graft applied after excision on dorsum of hand

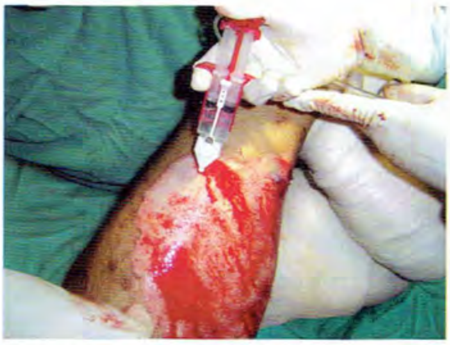

Fig. 15.5C: Hemostasis being achieved with fibrin sealant

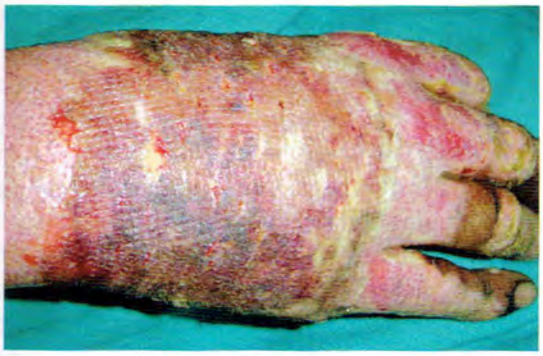

Fig. 15.4D: Postoperative picture showing complete take of graft on healthy bed

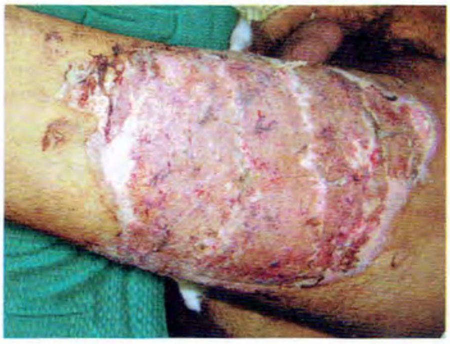

Fig. 15.5D: 2 weeks postoperative picture showing completely healed area

uses pinch technique, in which the skin adjacent to the wound is pinched to lift burnt area from the unburnt area as a skin fold and the excision is performed by Watson's skin grafting knife.

For harvesting graft from uneven areas like chest wall and scalp, it is advisable to inject saline in subcutaneous tissues.

Blood Loss during Excision

Operative blood loss during excision can be calculated by following formulae:

a. By measurement of pre- and postoperative hematocrit value:

Blood loss in ml =

$$\frac{(\text{Preoperative RBCV}) + (\text{T} \times \text{RBCV}) - (\text{Postoperative RBCV})}{\text{Postoperative hematocrit} \times 0.01}$$

Where:

RBCV= red blood cell volume

Preoperative RBCV (ml) = Body weight (kg) × 80 (ml/Kg) × [preoperative hematocrit (%) + 0.01]

Postoperative RBCV (ml) = Body weight (kg) × 80 (ml/Kg) × [postoperative hematocrit (%) +0.01]

T × RBCV = Total ml. whole blood administered during surgery × 0.03

Desai et al[18] have shown that

i. Average amount of blood loss for more than 30% TBSA burns when excised in the first 24 hours after injury was 0.41 ± 0.06 ml/cm^2

ii. Average amount of blood loss for more than 30% TBSA burns when excised between 2 and 16 days after injury was 0.75 ± 0.04 ml/cm^2

iii. Average amount of blood loss for more than 30% TBSA burns when excised more than 16 days after injury was 0.49 ± 0.07 ml/cm^2

b. *By measurement of pre- and postoperative hemoglobin value*: This formula implies that decrease in hemoglobin is due to intraoperative blood loss along with hemodilution because of large volume of intraoperative crystalloid transfusion.

Blood loss in ml =

EBV × [(preoperative Hb – postoperative Hb)/ average Hb] + Tx

Where:

EBV= estimated blood volume (70 ml/ Kg of body weight)

Preoperative Hb = preoperative hemoglobin in
Postoperative Hb = hemoglobin in g/L at 2
postoperatively

Average Hb = the mean of preoperative and posto
hemoglobin in g/L

Tx = Total intraoperative and 24-hours posto
transfusion volume received in ml.

Using this formula, Cartotto et al[25] calculated t
loss (mean ± SD) to be 211± 166 ml per % TBSA

Blood loss can be minimized by application o
epinephrine with or without thrombin to excised
and/or donor sites; the subcutaneous infiltra
vasoconstrictors such as epinephrine, phenylep
vasopressin at donor and/or excision sites; the admin
of systemic vasopressin; controlled intraop
hypotension; performing excision with laser; an
limb tourniquets.[25] Locally reacting fibrin sealants lik
are now available for use over such excised areas fo
of bleeding (Figs 15.3D and 15.5D) However, the
uniform, complete and consistent approach to
intraoperative blood loss. From time to time, conce
also been raised over assessing viability of recipi
when tourniquet is used or subeschar epinephrine
to reduce blood loss. This is because such methods i
with bleeding from the viable tissues left after exci

To reduce blood loss from donor area and bur
(to be excised) where tourniquet application is not
epinephrine solution 1:5,00,000 may be inf
subcutaneously until they become firm (i.e. tum
After harvesting graft, epinephrine saline s
(1:2,00,000) soaked gauze may be used topically.

To reduce blood loss during excision from t
areas over extremities, tourniquet is inflated 100
above systolic pressure. After excision is complet
epinephrine solution may be used for 10 minute
release of the tourniquet. After release of the tourni
limb is again wrapped in epinephrine solution soak
for another 5 minutes.

For assessment of viability of the tissues after
under tourniquet and epinephrine solution, the ap
of the tissues left is observed: the dermis must b
white, with no hemorrhagic staining; minor vessel
wound surface must be patent; the fat must be pale
firm, and moist (dry or golden brown fat is unheal
the excised wound rapidly becomes hyperemic, ev
tourniquet control. In particular, shortly after what
to be adequate excision, the white dermis and pal
fat appear to develop hemorrhagic staining. Altho

usually represents re-perfusion, it can easily be confused with the staining that characterizes thermally injured tissue. Thus, there may be a tendency to unnecessarily re-excise the area. Hence, the level of excision is most easily determined with the initial passes of the debriding blade and should not be altered thereafter unless there is convincing evidence of inadequate excision (e.g., tissue fails to re-perfuse, remains dry or contains thrombosed vessels on the wound surface).[25]

The order of excision: In cases of extensive burns, the first aim of excision should be towards coverage of the area (large and easily excisable areas e.g. trunk) rather than towards functional recovery (excision of small and difficult to excise areas like hand).

Advantages

As compared to en bloc excision, tangential excision produces less contour problems and better cosmetic result. This technique results in better texture of grafted skin and greater sensitivity.

Complications

The immediate complication is failure of graft 'take' which may be due to inadequate excision, infection, hematoma and lack of proper immobilization.

Problems

1. Cyst formation, probably as a result of blockade of the ducts of adnexal glands.
2. Overhanging skin graft margin.
3. Pinhead dark spots from inspissated secretions which disappears after meticulous cleaning.
4. Epithelial bridging showing hair follicle at the undersurface of the epithelium.[16]

Limitations: Paucity of graft donor area in extensive burn is taken care by use of allograft, homograft or synthetic skin temporarily till cultured auto-keratinocytes are available. Intermingled xeno/homograft and autograft may be used in absence of keratinocyte culture facilities[26].

EN BLOC EXCISION

Excision En Bloc (Figs 15.6A to 15.7D) is indicated for third degree burns and involves removing the burnt skin and all underlying burnt tissues (that may include fat, deep fascia and burnt muscle) en bloc till healthy bleeding tissue. This can be done with scalpel, electric cautery or laser scalpel.[16] When excision is done up to the level of deep fascia (sometimes including muscle fascia), it is called fascial excision (Figs 15.6A and B).

In fascial excision, the skin, subcutaneous tissue and deep fascia is incised by knife and scissors. Traction is applied over the tissue to be excised and lifted from the plane deep to the deep fascia and burnt tissue is excised en block. If muscle is involved, it is debrided thoroughly. During en bloc excision (Figs 15.7A to D) fewer and larger vessels are encountered. To reduce blood loss, these vessels should be divided after ligating or cautery application. To ensure good graft 'take', perfect hemostasis is achieved before covering the area with skin graft.

Advantages

Blood vessels are easily identified at the level of deep fascia and hence, tackled easily to reduce blood loss. Graft 'take' is usually better than that on fat exposed after excision of burn involving full thickness of dermis.

Problems

1. Excision of subcutaneous tissues alters body contours (aesthetically poor result), results in poor texture of grafted skin and poor sensitivity. Hence, whenever possible, fat should be preserved over face, hands, dorsum of foot and areas around the joints. In females one should attempt to preserve as much mammary parenchyma as possible.

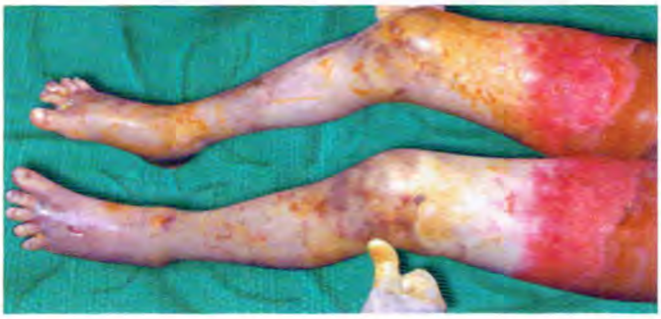

Fig. 15.6A: Third degree burn of both lower extremities

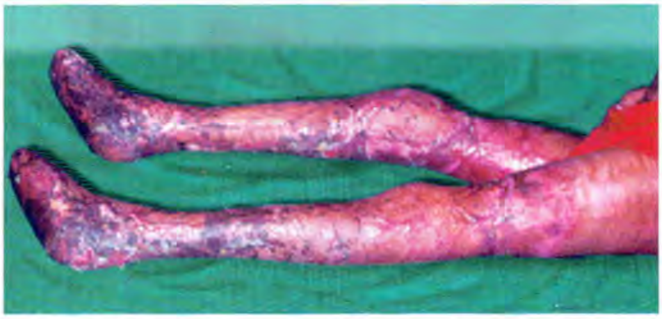

Fig. 15.6B: Result after fascial excision and split skin grafting

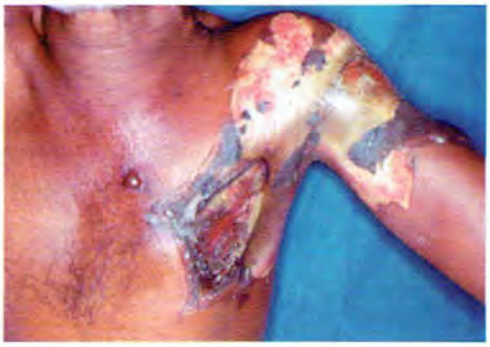

Fig. 15.7A: Third degree burn of chest and right upper extremity

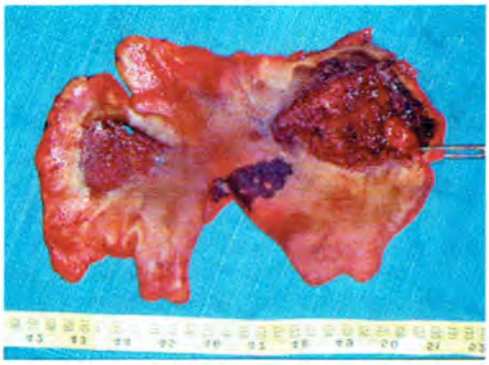

Fig. 15.7B: Excised tissue

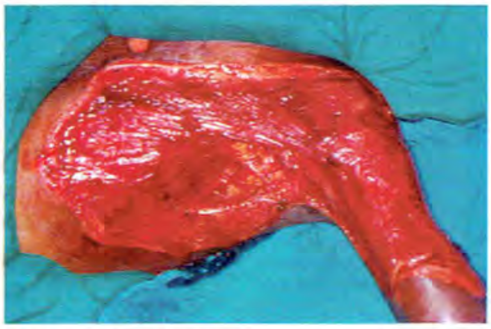

Fig. 15.7C: Viable tissue following en bloc excision

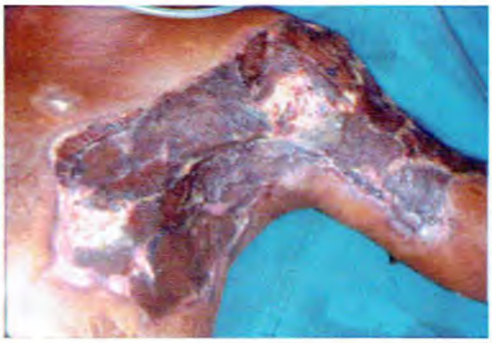

Fig. 15.7D: Result after en bloc excision of burnt tissue including superficial muscle

2. While excising over tendoachillies and tendons dorsum of hand and foot, the importance of stayin correct plane can not be overemphasized in orde avoid exposure of bare tendons.

Whatever technique is used, there is no substitute experience of the surgeon and good facilities in a ce where routine excisional surgery is performed.[4]

Immunological benefits of wound excision and clos Wound excision and closure causes decrease proinflammatory mediators, decreased endotoxemia restoration of contact hypersensitivity. It has been sh that burn injury inhibits cytotoxic T lymphocyte (C function (which is involved in antigen recognition, proces and elimination) in a size dependent manner and total wo excision significantly improves both CTL function survival after injury. Therefore, it has been suggested early excision may help to restore host immunocompete and may decrease the infectious complications of ther injury.[27]

Wound Debridement: In cases where burn excision split skin grafting is not done due to medical or o reason(s), excision of dead tissue i.e. wound debriden (Escharectomy, Slough excision) is done, if nee repeatedly at 48-72 hours intervals followed by repe dressings till healthy granulation tissue (fit for split grafting) appears.

POSTOPERATIVE MANAGEMENT

In the immediate postoperative period, hemoglobin, P serum electrolytes level is checked to assess blood loss appropriate action is taken as per report. Hourly vital si and urine output is measured and intravenous flui administered as per requirement. Antibiotics, as per cul and sensitivity report, analgesic and supportive treatme instituted. Depending on gut function, oral feeding is sta after 6 hours or later.

Depending on wound condition, dressing is done day 3-5 and then, on alternate days. Donor area heals in 14 days. Physiotherapy is started after 7 days. Any splir required, is delivered at the time of discharge or du follow up visits.

SUMMARY

- Wound excision and skin grafting is an establis method of treatment for deep dermal, indetermin depth and deep burns.
- Technique-wise, excisional surgery is classifie tangential or en bloc excision. Based on the tim

excision, it may be classified as acute (within 24 hours), early (within 3-5 days) or delayed (within 6-16 days) excision.

- Excisional surgery has led to reduced overall morbidity significantly and to some extent, burn wound sepsis and mortality. It has also led to reduced cost of treatment, reduced wound related pain, better functional result, early rehabilitation and reduced long term morbidity with reduced time away from work.
- As compared to en bloc excision, tangential excision produces less contour problems and better cosmetic result. This technique results in better texture of grafted skin and greater sensitivity.
- Experience of the surgeon and facilities available at the center where excision is done is important. Less than 20% TBSA excision is safe. If treated with excision and grafting 20-40% TBSA burn patients appear to have fewer infectious wound complications and a shorter hospital stay.
- A significant increase in blood loss should be expected with wound excisions after 5 to 6 days as infection and inflammation of the viable tissue below eschar might increase its vascularity. Clotting abnormalities at this stage can also be a major problem.
- Chances of sepsis increase if excision is delayed by more than 5 days.
- By early excision and grafting, full functional recovery may be achieved within 2 weeks, whereas in the conventional method, de-sloughing alone requires 3 weeks and the shortest possible time for full healing is therefore about 5 weeks.
- Scarring (hypertrophy, contractures) is less if wound is closed early and there is reduction in the need for reconstructive procedures.

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Chapter

16

Anesthesia for the Thermally Injur

PK

INTRODUCTION

The first written documentation describing the treatment of thermally injured dates back to almost four millennia.[1] However, it is only in the last 60 years that any significant improvements in morbidity and survival have occurred. In the past, up to 20% deaths were due to the effects of hypovolemia, contributing to early mortality following burns. Currently, most deaths occur several weeks after injury and are due to multiple organ failure, often in association with sepsis. More people are surviving major burns than ever before and the percentage of total body surface area (% TBSA) burn that is lethal to half the victims is now in the region of 70% in the population up to the age of 45 years.[2] Improved survival rates are due to the adoption of early tangential excision of the burn wound, techniques of infection control, technological advances in cardiac, respiratory and renal support in the intensive care unit and an overall greater aggressive approach to the management of this injury by intensivists.

The anesthetist is an essential member of the burn management team possessing both the skills as well as the knowledge required for the treatment of the burn injured at all stages. The need for specialized burn anesthesia is obvious as these patients undergo many operations, sometimes within a short period of time. They need multiple blood transfusions following extensive blood loss during early excisional surgery. A burn victim needs an integrated approach by the burn team to achieve minimal morbidity and mortality.

PATHOPHYSIOLOGY

It is important to understand the early pathophys
changes that occur with thermal injury as successful
management relies on the response to resuscitation.
injury produces many of the physiological chan
are seen in any major trauma but are more profo
longer lasting.

Cardiovascular Changes

After a burn injury, especially over 20% total bod
area (TBSA) burn, there is a loss of capillary int
both normal and burned tissues, resulting in a loss o
volume into the interstitial space and the burn wounc
this resuscitation phase, lasting 72 hours, aggress
replacement is essential to maintain the perfusio
vital organs. In addition, adequate intravenous ana
administered not only for the comfort of the pa
also because the pain alters the vital signs that are
to monitor resuscitation.

The fluid shifts in the early post burn period
the cardiac output independent of the status of intra
volume. Reduced cardiac contractility resul
circulating humoral factors, reduced responsive
decreased coronary blood flow. The magnitude o
impairment is larger in the older patients. Systemic
resistance also increases, coincident with the dec
cardiac output.

After successful resuscitation in the first 24-
after a burn, the myriad of inflammatory media
cytokines released change the cardiovascular resp

the classical systemic inflammatory response syndrome (SIRS). This syndrome is manifested by an increased cardiac output and reduced systemic vascular resistance and reflects a marked increase in the metabolic rate. The consequences of this are important. These systemic inflammatory response syndrome manifestations are identical to those seen in sepsis and care must be taken to avoid antibiotic use in the absence of other evidence of systemic infection. In addition, this syndrome is the prime cause of hypermetabolism seen in a severely burnt patient.

Respiratory System Changes

Upper airway injuries result from inhalation of superheated air or steam and toxic compounds found in smoke. Brief exposure of the epiglottis or larynx to either dry air at 300°C or steam at 100°C leads to massive edema and rapid airway obstruction. In children, this may result in macroglossia, epiglottitis and laryngotracheal bronchitis. Even patients with a mild upper airway injury are at risk for development of progressive airway obstruction as tissue edema develops. The clinical signs (Table 16.1) that should alert the clinician to the possibility of upper airway burns are facial burns, singed nasal vibrissae, bronchorrhea and sooty sputum (Fig. 16.1). If severe, patient may appear dyspneic, use accessory muscles of respiration and sit upright. Hoarseness and dysphagia are common. Flow-volume curves and nasopharyngoscopy may aid the initial diagnosis of upper airway burns and are particularly useful in assessing trends in airway impairment. If an inhalational injury is suspected, it is vital that the airway be secured at the earliest possible time. Intubation is mandatory in a large burn with an inhalational injury as resuscitation may cause massive swelling and jeopardize the patency of the airway (Fig. 16.2). In fact, any delay may make a subsequent attempt at intubation physically impossible due to loss of identifiable landmarks as a result of severe edema of all the structures above the larynx. It may be wise to avoid performing a tracheostomy on these patients as this procedure is associated with a high mortality[4] (Fig. 16.3).

Table 16.1: Clues to inhalational injury

- Exposure in a closed-space
- Loss of consciousness
- Nasal, oral or facial burns
- Singed nasal vibrissae
- Carbonaceous sputum
- Hoarseness or stridor
- Upper airway changes on bronchoscopy
- Flow–volume curves
- Chest radiograph showing pulmonary edema

Hypermetabolic state manifests clinically as a persistent tachycardia (despite adequate resuscitation), hyperpnea and marked muscle wasting.

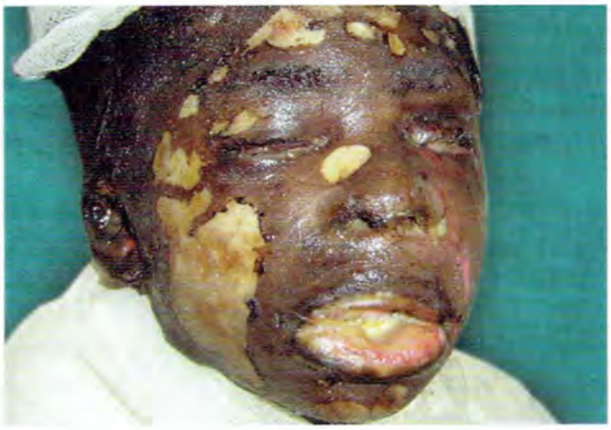

Fig. 16.1: Patient with face burns along with inhalation injury

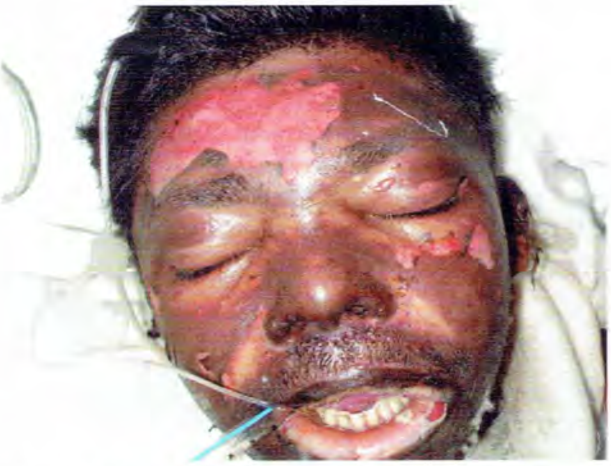

Fig. 16.2: Patient with inhalation injury requiring intubation for mechanical ventilation

The oropharynx and upper airway are efficient heat exchangers. Therefore, thermal burns to the lower airway are rare unless steam or chemicals have been inhaled. The clinical signs described for upper airway injury are also applicable to lower airway injuries. In addition, the patient may give a history of being confined in a smoke-filled, closed space and may exhibit raised levels of carboxy-hemoglobin.

If the thorax is burned, chest wall compliance may be decreased by edema, by the formation of an inelastic eschar and possibly by the application of tight bandages. Lung compliance and functional residual capacity may also be reduced. Minute ventilation and the alveolar/arterial gradient for oxygen increase, mainly because of edema and reduced

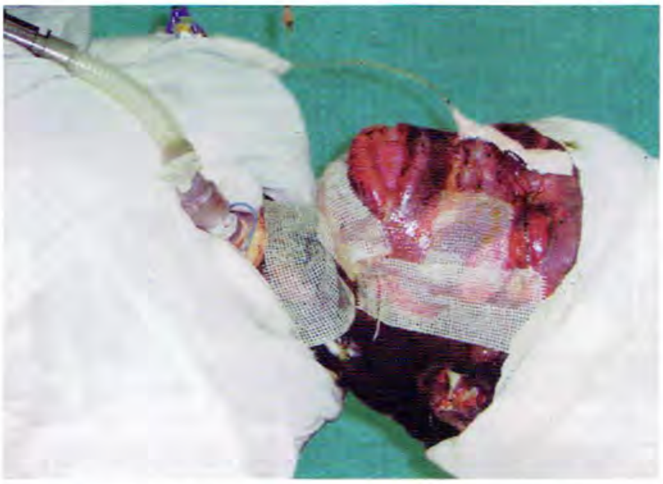

Fig. 16.3: Patient with severe inhalation injury requiring tracheostomy for ventilation

cardiac output. Hypoxia coupled with increased work of breathing may exhaust the patient. These considerations reinforce the necessity for early tracheal intubation, high concentrations of oxygen and controlled ventilation.

Metabolic Changes

The burn injury results in a massive catecholamine output. The outcome is a hypermetabolic state characterized by protein catabolism, urea genesis, liploysis and gluconeogenesis. In patients with burns of more than 50% of the total body surface area, the basal metabolic rate may be doubled.

The loss of skin and the increased metabolic rate impaire the thermoregulation of the patient who responds by increasing his core temperature to 38.5°C. This persistent pyrexia may mask the early signs of sepsis which is the most common cause of death once the patient has reached a burns unit. A high index of clinical suspicion, supported by hematological and bacteriological surveillance, is essential and aggressive antibiotic therapy must be initiated early.

Ambient temperature has an important effect on the metabolic rate in burn patients. A study of patients with a mean burn size of 44% TBSA showed that patients at thermoneutral ambient temperature (28-32°C) had metabolic rates 1.5 times those of unburned controls. However, when ambient temperature was decreased to 22-28°C, the metabolic rate increased in proportion to burn size. Thus, ambient temperature less than thermoneutral range should be avoided, whether in the burn unit or in the operating room, to minimize further increase in metabolic rate.

The increase in metabolic rate has implic
nutritional support in the post burn period. Ea
decreases muscle catabolism and may reduc
translocation through the intestinal mucosa. Ade
control, alleviation of anxiety, a thermoneutral e
and an aggressive treatment of infection are all
steps in limiting catecholamine secretions
hypermetabolism which diverts calories from t
tissues.

Immunologic Changes

Sepsis and multiple-organ failure are a major cau
among burned patients. Large areas of dead tissue
colonized with bacteria and the burned patient is
fight infection as a result of impaired white
function and reduced levels of fibron
gammaglobulins.

ANESTHETIC MANAGEMENT

Convalescence from a thermal injury can be di
three phases. The anesthetic requirements durin
phases are different and will be discussed separ
is the resuscitation phase, which starts at the time
sustains the burn. This phase deals with mainten
airway, acute fluid replacement and cardi
stabilization and analgesia (Table 16.2). All anest
be familiar with the acute resuscitation of victi
burns. Hemodynamic stability and adequate u
should be established within 72 hours after inj
resuscitation phase is now complete. This is f
the hypermetabolic stabilization phase (when
are used to cover the defects). This phase may l
days or more. Finally, the third phase, i.e. the rec
phase deals with the restoration of form and
damaged tissues and the release of contracture

Resuscitation Phase

The role of an anesthesiologist in the acute resu
a severely burnt patient varies from institution to

Table 16.2: Problems encountered by the anest
in the resuscitation phase

- Airway management
- Cardiovascular stabilization
- Fluid replacement
- Analgesia
- Life-saving surgical interventions.

The special skills of the anesthetist in airway management, fluid management and percutaneous, intravenous and arterial catheterization may be required. The detailed discussion of the management during this phase is beyond the scope of this chapter and has been discussed elsewhere.

Anesthesia during resuscitation phase should be provided only for life-saving procedures. Common surgical problems which may require anesthesia are usually injuries associated with burns, for example, amputation of non-viable limb, exploratory laparotomy or thoracotomy for penetrating injuries, etc. All else should be postponed till the patient has been resuscitated and stabilized. In the presence of injuries like fractures, abdominal or thoracic trauma which are commonly associated with blood loss, blood replacement may be required in addition to resuscitation fluids. Ketamine may be the safest agent for anesthesia during this period and may be used in combination with a non-depolarizing neuromuscular blocking agent (NMBA) if relaxation is required. Suxamethonium may be used to facilitate intubation from the time of injury to 48 hours after injury but should be carefully avoided thereafter. Increased oxygen demand, tachycardia and decreased compliance may make mechanical ventilation (MV) mandatory both, preoperatively and postoperatively. Adequate analgesia must be provided in the perioperative period and great care must be taken to ensure continued adequate resuscitation during anesthesia.

Hypermetabolic Stabilization Phase

Some of the commonly performed operations during this phase include primary excision and skin grafting, dressings and debridement.

The pathophysiological state of the patient presenting for surgery and anesthesia varies depending on the percentage of TBSA burnt, location of burns, age of the patient, time elapsed since initial injury and adequacy of the treatment received in the resuscitation phase[5] (Table 16.3). Blood loss due to increased wound vascularity, hypothermia and plasma potassium levels are the predominant considerations during surgery on the burn patients in this phase.

Table 16.3: Factors modifying the pathophysiological state of the patient

- % TBSA burnt
- Location of burns
- Age of the patient
- Time elapsed since initial injury
- Adequacy of the treatment received in the resuscitation phase

Preoperative Assessment and Preparation (Table 16.4)

Successful anesthesia for excision and grafting of a burn patient requires planning. Because burn patients often undergo many operative procedures, it is important for the anesthetist to establish good rapport with the patient and explain all movements and procedures in advance, especially if they may be painful.

A brief history should be obtained, either from the patient or from the progress notes. The cause of the burn should be ascertained. Electrical burns may cause myocardial damage. Chemical burns are associated with a high incidence of renal or hepatic damage. Exposure to toxic fumes or concurrent inhalational injury may interfere with respiratory function. Past medical history, allergies, recent medications, previous surgery and previous anesthetics should be reviewed.

Physical examination may be limited by dressings, dyskinesia or pain. The extent of the burn and other injures

Table 16.4: Considerations in preoperative evaluation/during preoperative visit of the burned patient

- Reassure the patient; establish rapport; explain the procedure in advance.
- Obtain history of the patient
  - Ascertain the cause of burn (chemical, electrical, inhalational etc.)
  - Review any past history of relevance.
- Perform physical examination
  - Record the extent of burn and other injuries
  - Assess current IV access and whether the current site needs a change
  - Assess the airway and predict if a difficult intubation is likely
  - Assess for adequacy of resuscitation (mental status, skin color, capillary refill, hematocrit, oxygen saturation, urine output) and correct any deficiency.
  - Evaluate cardiovascular system (ECG, BP, CVP, Pulmonary artery catheterization, if indicated)
  - Ascertain the respiratory status (X-ray chest, ABG)
  - Look for evidence of any coagulation abnormality (Hb/ Hematocrit, electrolytes, coagulation profile)
  - Any evidence of acute renal failure (blood urea, serum creatinine, serum electrolytes, urine output, urine osmolality).
- Interact with the burn surgeon (nature and extent of excision, amount of blood loss, operative time)
- Arrange for the estimated blood loss
- Premedication
  - Provide adequate analgesia
  - Brief period of fasting
- Take a decision about the monitoring and care to be provided during transport.
- Consider antithrombotic prophylaxis.
- Record any additional point(s) of relevance.

should be ascertained from the recorded notes. The respiratory and cardiovascular system should be examined as in any other assessment. The sites of possible venous access should be evaluated as these may be limited and difficult to find. The assessment of the airway and predicting, if a difficult airway is likely, is important in these patients at this stage. Finally, adequacy of resuscitation is observed/checked. Simple parameters like assessment of mental status, skin color, capillary refill, hematocrit values, oxygen saturation and urine output should be employed fully. Noninvasive blood pressure measurement and electrocardiographic (ECG) monitoring should be a routine, but unusual sites may be required for the cuff and electrodes. Placement of a sterile cuff over burned areas is acceptable or an unsterile cuff may be placed over a thin occlusive dressing. Invasive arterial and central venous pressure monitoring may be helpful, particularly for patients with extensive burns or an inhalational injury. However, these carry significant risk of infection.

Of all the parameters available, urine output is probably the most useful. If resuscitation is adequate, the urine output should be at least 0.5 ml/kg body weight/hour and the osmolarity of the urine in the range of 600 to 1000 mosm/l. In the presence of myoglobinuria, mannitol may be given to promote diuresis. In these patients the urine output is no longer a reliable guide to the adequacy of resuscitation. If resuscitation is inadequate, this is the time to take corrective measures.

Respiratory status of the patient should be assessed carefully during the preoperative assessment and strategy to deal with the findings planned accordingly. A patient with burns around the airway (e.g. singed nasal hair) should be intubated as early as possible. Direct inhalational/thermal injury and fluid resuscitation may make delayed intubation more difficult secondary to upper airway edema, as discussed earlier. Physiologic derangements due to injury to lower airways may include pulmonary edema and acute respiratory distress syndrome (ARDS). Additionally, burn patients can be severely hypermetabolic, with corresponding increased carbon dioxide production. These patients may have high peak inspiratory pressures (PIPs) and high minute ventilation requirements. High level of positive end-expiratory pressure (PEEP) may be helpful in these patients. Other possible effects of severe burns include: decreased lung and chest wall compliance, decreased functional residual capacity (FRC), increased alveolar-arterial oxygen gradient, increased carboxy hemoglobinuria and increased methemoglobinuria. Chest radiograph and arterial blood gases (ABG) should be asked for to evaluate the progress of patient's condition. Septicemia and chest infections generally associated with respiratory failure and indications for controlled mechanical ventilation.

The variations of urea and electrolytes in blood sh be corrected preoperatively. An element of renal fai associated with inadequate resuscitation may be pres The blood sugar is usually elevated and may cause an osm diuresis with a concomitant loss of electroly Coagulopathies may result directly from the burn inj especially in the 2nd week post burn, as well as from r replacement of blood loss during operative procedure should be monitored closely. Coagulation profile, hemogr hematocrit and electrolyte reports should be analyze monitor the condition of the patient. The hematocrit, i else is normal, indicates the level of hydration.

Preoperative anemia is, in part, dilutional in the e days, but levels below 8 gm/dl should be corrected be surgery. The attention to anticipated amount of blood should be given at this visit. Postoperative oozing i dressings can be considerable and this loss should als taken into account when ordering blood to maintain hemoglobin concentration above 10 gm/dl. It is impor (during this preoperative visit) for the surgeon anesthetist to decide collectively, about the nature and ex of the excision, the amount of blood loss anticipated the anticipated operative time in view of the patie condition. The endpoints for surgical excision in large bu are: (i) Operative time of 2-3 hours; (ii) core temperatur 35°C; or (iii) blood loss of 10 units of packed red bl cells. The violation of any of these parameters inv coagulopathy and increasing problems with hemostasis vital signs stability.

Preoperative fasting guidelines have been modifie burn patients for several reasons. Achieving adequate cal intake in patients with major burns is difficult and nutriti support is frequently supplemented with nasogastric t feedings. Thus, minimizing the fasting period bef operation is beneficial. It is advisable not to disconti enteral feeding before operation in patients who are alre intubated with cuffed endotracheal tubes; in non-intuba patients, feeding should be stopped 2 hours prior to surge This improves preoperative nutrition without appare increasing the risk of aspiration.

Premedication should be ordered; an opioid analg like morphine or pethidine along with promethazine sho be used. An anti-sialagogue such as atropine glycopyrrolate may also be included in the premedicati

The effect of the premedicant analgesic should be noted because these patients may be unduly sensitive or apparently resistant to analgesics. If all that is needed is an anxiolytic, then a benzodiazepine such as lorazepam may be used. The property of anti-recall, which is profound with lorazepam, is very beneficial in a patient who undergoes many procedures. Ketamine continues to have a role in premedication for children via the oral or rectal routes.

Antithrombotic prophylaxis should be considered, balancing the hypercoagulable state of the major burn with the risk of disseminated intravascular coagulation (DIC) and the potential for deep vein thrombosis in any non-ambulatory patient against the risk of hematoma formation under skin grafts or skin substitute.

Preoperative visit is also the time to plan the ways to transport the patient (depending on the condition) from ward to the operating room. The patient may be brought to the operating room on the ward bed as any movement to and from a transport trolley causes unnecessary pain. For patients with severe ARDS, transportation may pose formidable challenges with regard to ventilation. Cardiopulmonary monitoring must be continued during transport; the ventilation system used in transport must be capable of delivering high minute volumes, PEEP and inspiratory pressures. These requirements may not be satisfied by standard bag-valve systems and may require a high-quality transport monitor.

The following conditions may be considered as contraindications[7] for surgery:

- Inadequate resuscitation;
- Abnormal electrolytes;
- Respiratory or metabolic acidosis;
- Abnormal clotting studies;
- Incipient septicemia;
- Unexplained rapid temperature changes;
- Changed consciousness level;
- Instability in respiratory or cardiovascular parameters.

Intraoperative Period

Whilst there is no single technique that may be regarded as ideal, a number of general principles apply (Table 16.5). Skin-grafting or free-tissue transfer must not be jeopardized by poor perfusion during or after anesthesia. The use of hypotensive techniques, smooth balanced anesthesia and excellent analgesia with good fluid/blood replacement contribute to a successful outcome and avoid anesthesia being blamed for poor surgical outcome. Regional techniques are rarely feasible, given the multiple surgical sites for harvesting and skin grafting. Laryngeal mask airways (LMAs), in general, are not recommended for major burns, given the potential for significant fluid resuscitation and subsequent airway edema, as well as the frequent repositioning of patient intraoperatively. LMAs, however, are invaluable for shorter procedures, avoiding repetitive

Table 16.5: Anesthetic consideration for excision and grafting of major burns

- **Premedication**
  - Provide adequate analgesia
  - Brief period of fasting
- **Vascular access**
  - Establish large-bore IV access
  - Consider invasive monitoring
- **Induction**
  - Volume resuscitated: Propofol /Thiopentone
  - Volume depleted: Ketamine
- **Securing the airway**
  - Consider alternatives to direct laryngoscopy
  - Consider awake intubation
- **Ventilation**
  - Ventilation requirement, due to:
    - Hypermetabolism
    - Parenteral alimentation
  - Smoke inhalation / circumferential burns / ARDS may necessitate ICU-type ventilator
- **Fluid and blood**
  - Blood loss can be extensive
  - Check coagulation profile
  - N-saline / Ringer lactate @ 8-10 ml/kg/hour
  - Keep urine output 0.5-1 ml/kg/hour
  - Continue parenteral nutrition
- **Temperature regulation**
  - Warm all fluids and keep operation theater warm
  - Humidify gases
  - Warming blanket/reflective head cover
- **Selection of anesthetic agent**
  - Include opioids with all techniques
  - Catecholamine (endogenous and exogenous) levels are increased: choose/use compatible inhalational agents
- **Muscle relaxants**
  - Avoid succinylcholine
  - Resistance to NMBAs (non-depolarizing muscle relaxant)
- **Recovery/Postoperative state**
  - Opioid requirements are large
  - Keep the patient warm
  - Check coagulation profile
  - Continue cardiopulmonary monitoring
  - Oxygen administration
  - Consider initiating enteral nutrition as early as possible

Adopted and modified from: MacLennan N, Heimbach DM and Cullen BF. Anesthesia for major thermal injury. Anaesthesiology 1998; 89: 749-70 laryngeal instrumentation and for their ability to maintain the difficult airway while deepening the anesthetic or as an aid to intubation with or without the fiberoptic bronchoscope[8](Fig. 16.4).

Induction of Anesthesia

The patient should be brought to the induction room on the ward bed (if facilities exist), as any movement to and from a transport trolley causes unnecessary pain. Induction of anesthesia can be performed on the bed and the patient transferred to the operating table once profound analgesia has been obtained. Non-invasive monitoring should be placed in site prior to induction of anesthesia (Fig. 16.5). A second anesthetist is helpful to assist in induction. A surgeon should be available in the induction room. He may be called upon to perform a cut-down on a vein if percutaneous venous access is not possible. Securing venous access is a prerequisite to burn excision and grafting procedures. This may be established the night before surgery to ensure adequate hydration. For most cases, a minimum of two large-bore peripheral intravenous (IV) lines or one peripheral and one central line should be used. These may be inserted under local anesthesia with xylocaine and used for induction. Insertion of large-bore cannulae can be performed under general anesthesia. The positioning of both IV access and monitoring should be discussed with the surgeon with respect to the operative site.

Pre-oxygenation is advisable, because of the high metabolic rate, tachycardia and increased oxygen needs. If the trachea is assessed as being easy to intubate and patient is adequately volume-resuscitated, fentanyl at 3-5 mg/kg body weight may be given by slow IV injection. This may be followed with an induction dose of propofol (1-2 mg/kg).[9] This technique allows for rapid recovery and minimizes postoperative cognitive disturbances. Alternatively, thiopentone (3-5 mg/kg) may also be used. Xylocaine (1-1.5 mg/kg) may be given prior to intubation to depress the cardiac response to intubation and enhance the potency of fentanyl. If the patient is intravascularly volume-depleted, ketamine (1-3 mg/kg) is recommended. Patients with extensive burns (>30% TBSA) develop a decreased sensitivity to non-depolarizing neuromuscular blocking agent (NMBAs); therefore, 1.5 times the usual intubating dose of NMBAs is recommended: vecuronium (1.5 mg/kg), pancuronium (0.15 mg/kg), or rocuronium (1.5 mg/kg). If rapid sequence induction is indicated, high dose rocuronium (2 mg/kg) can be used. Succinylcholine is contrair during this phase (i.e. when injury is >48 hours old) l of potentially lethal efflux of potassium ions from This abnormal response probably occurs because th muscle membrane, rather, than just the myoneural j becomes a receptor.

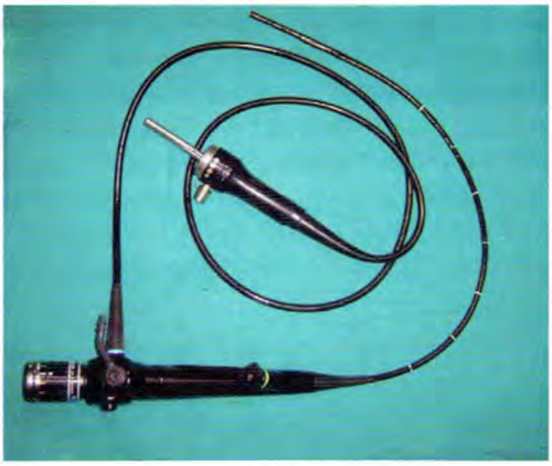

Fig. 16.4: Fiberoptic set

Fig. 16.5: Anesthesia work station with attached noninv monitors

In case, a difficult intubation is anticipated, alt plans should be sought for. As mentioned tracheostomy is not recommended, being associat higher mortality. Most injuries leading to difficult int will have burns around the face and neck which ar to be infected. If a tracheostomy is performed, it m to septicemia, mediastinitis or chest infection, ult causing the demise of the patient. Facial burns, wit area on the face, may make placing a face mask difficult on the face. A sterile Gamgee pad, with a hole in the area of nose and oral aperture, may be used to cover the raw area and the face mask can be kept on the pad. The other methods of intubation which can be considered include inhalational induction followed by blind nasal intubation, awake fiberoptic intubation (Figs 16.6A to C), cricothyroidotomy with jet ventilation, cricothyroid puncture and retrograde cannulation or percutaneous tracheostomy. The LMA has proved its worth and is an acceptable alternative method to try for shorter procedures (Fig. 16.7). Securing the endotracheal tube (ETT) can be difficult in the presence of facial burns (Fig. 16.8). Alternatives to taping the ETT include suturing the tube to the lip[10] or using a circumferential tie around the patient's head.

Maintenance of Anesthesia

Controlled ventilation, facilitated with muscle relaxants, is the method of choice for all but the shortest procedures, i.e. dressing changes. This technique reduces oxygen consumption and makes it possible to increase the minute volume to ensure that normal carbon dioxide levels are maintained in these highly catabolic patients. Physiologic derangements of the respiratory system (poor gaseous exchange and a low compliance) and a hypermetabolic state may require high inspiratory pressures, PEEP, and other ventilatory adjuncts to maintain oxygenation. Depending on equipment availability, an ICU ventilator may be necessary. The effect of positive pressure ventilation, PEEP and hypovolemia may summate to have a deleterious effect on the cardiac output and renal function and must be avoided by ensuring adequate intravascular volume and if required, by the use of inotropic agents.

Inhalational agents may be used to provide balanced anesthesia. Intraoperatively, burn surgeons commonly use epinephrine soaked sponges to decrease blood loss. Systemic absorption of epinephrine causes tachycardia and increases the probability of dysrhythmias; therefore, it is better to avoid halothane or desflurane. Isoflurane and sevoflurane are acceptable. The prolonged or repeated use of nitrous oxide may decrease erythropoesis by inhibition of vitamin B_{12}. This can be overcome by hydroxycobalamine injections.

Circumferential burns may require the patient to be moved during the procedure. An armoured tracheal tube and a lightweight co-axial breathing system, such as the Bain system may be preferred. The cornea should be

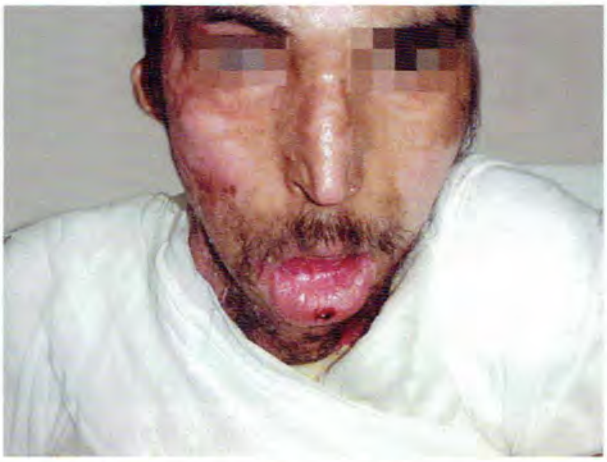

Fig. 16.6A: A patient with chronic post burn raw area with contracture of neck with difficult airway

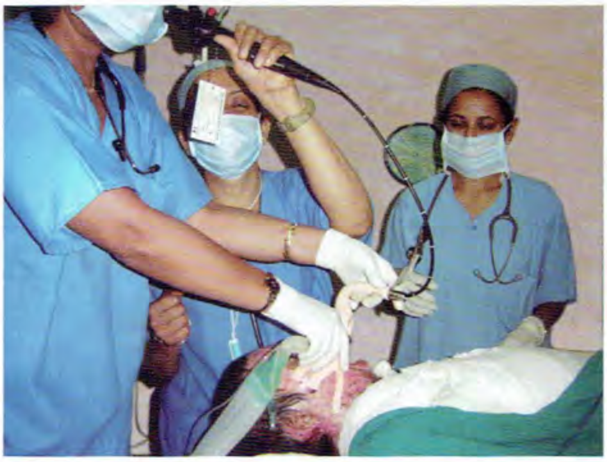

Fig. 16.6B: Fiberoptic bronchoscopy through intubating LMA being tried for the same patient through oral route because of pinched nostrils

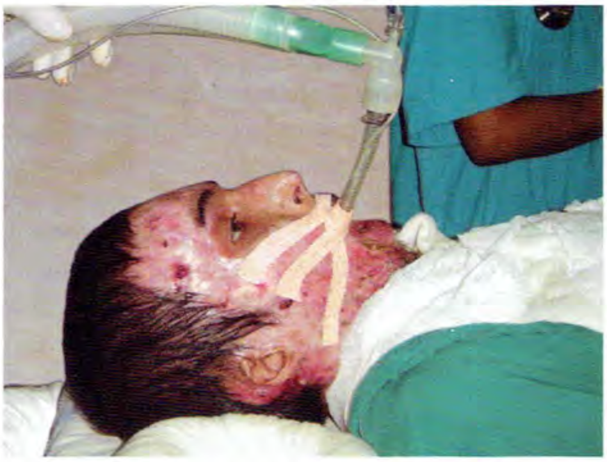

Fig. 16.6C: Same patient successfully intubated orally

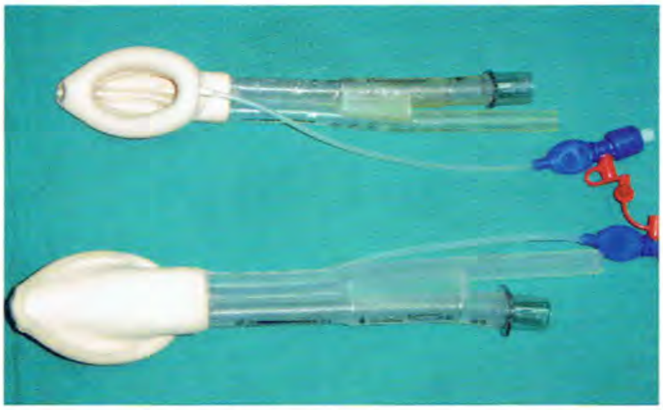

Fig. 16.7: LMA (Laryngeal mask airway)

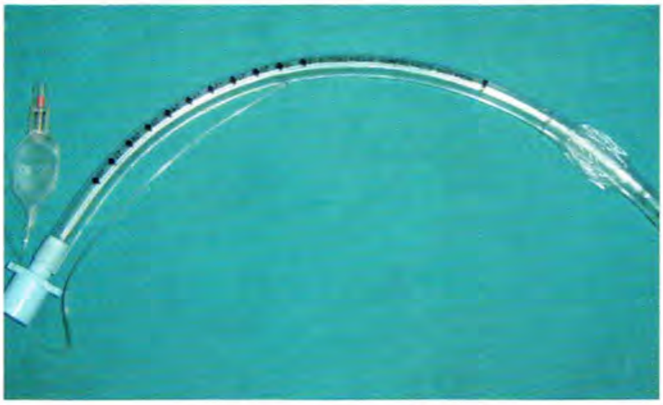

Fig. 16.8: Endotracheal tube

protected with eye ointment and pressure points should be padded. Facial burns may make eye closure difficult, and a temporary tarsorraphy may be required after prior discussion with the patient.

Adequate analgesia should be given during the intraoperative period and should continue in the postoperative state. It is humane to complete the dressing and place the patient on the ward bed before terminating anesthesia. Inadequate post-operative analgesia may make the patient restless; this in turn may provoke bleeding under the skin grafts (and their subsequent loss) or alternatively, displacement of the grafts. If large volume resuscitation has occurred intraoperatively, there is a possibility of clinically significant airway edema; use caution before extubating a patent airway.

Monitoring

Monitoring methods which are taken for granted in non-burn patients may be difficult in the burned patients. The blood pressure cuff may be applied to a lower both upper limbs are to be operated upon. The blo cuff may be rotated from one extremity to accommodate the surgery or an arterial cathet inserted. Surface electrodes may not adhere tincture of Benzoin has been applied, in w percutaneous needle electrode may be Transesophageal electrodes may be the method even though they are expensive. The pulse rate c be measured manually, by the arterial waveform tracing. The pulse oximeter is a useful moni indicates hemoglobin saturation as well as the pu suitable pulse for the sensor may be difficu Alternative sites include the earlobe and nasal s esophageal stethoscope can be used to monitor and breath sounds. Capillary refilling can be easil and the patient's color noted. These clinical ob may be all that is available in certain patients and c

Because of increased metabolic rate and carb production, increased alveolar ventilation may b Arterial Blood Gas analysis (ABG) must, the assessed early and frequently throughout the procedure. Constant monitoring of expired carb is very helpful during intraoperative management. one must be cognizant of the possibility of s differences between arterial and expired carbc values as a result of shunting and dead space ver patients with severe pulmonary injury.

All patients with major burns have a urin catheter in place. Adequate fluid replacement ensu urine output but a decreased output is a late sign of i replacement. Temperature control is a major pr heat loss can be enormous. Both core and p temperatures must be monitored. Heat loss is min warming all fluid, especially those used to prepa surgery. The thermoneutral temperature for thes is between 31°C and 33°C. This can be e uncomfortable for the working staff. A comp between 27°C and 30°C is usually made. Areas operated upon must be covered; overhead heaters a blankets should be used, especially on children; a covering the head may make a noticeable differenc peripheral temperature may be due to an inadequ volume if the core temperature is remaining stat

The most important feature of the intraoperat is monitoring and replacing patient's blood expected blood loss may be 100-200 ml/%BSA e grafted. Therefore, one must be familiar with the surgical approach to burn excision. During a tangential excision, a patient might loose three to five times more blood than during excision down to fascia. Intraoperative tourniquet use on burned extremities reduces overall blood loss and also decreases the rate of loss, making intraoperative hypovolemia less likely. Some centers use postoperative compression dressing after excision and grafting to reduce blood loss. Epinephrine (500 μg/l in normal saline) injected subcutaneously in both donor and excision sites are also used to reduce blood loss during excision and grafting procedures. Although high levels of catecholamines in the blood have been measured with this technique, complications such as dysrhythmias are uncommon. After burn injury, cardiovascular responses to catecholmines are attenuated because of a reduced affinity of the β-adrenergic receptors for ligands and decreased second messenger production. Blood loss is also quite dependent on the expertise of the surgical team. Hypotensive anesthesia has also been advocated as a means to decrease blood loss. Exact estimation of the blood loss is difficult because of bleeding into dressings, drops, sponges and packs, and on to the floor. Other indicators of circulating blood volume, such as urine output, central venous pressure, arterial pressure and shape of the arterial waveform, whenever possible, must be closely monitored. Blood loss should be replaced adequately. For patients without contraindications to hemodilution (e.g. coronary artery disease, anemia), it is often better to delay packed red blood cells transfusion until major blood loss is complete. If sudden decrease in blood pressure occurs during very rapid transfusion of blood (>150 ml/min), using calcium to counteract the chelating effect of citrate is considered. However, one should be cautious to avoid over transfusion especially in a small child or in an elderly. As the surgical site is superficial, there is not much third space loss.

Total parenteral nutrition should be continued throughout the procedure in order to avoid large changes in blood sugar levels which may occur when parenteral nutrition is constantly interrupted by surgical procedures. Difficulties are encountered due to the effects of stress response on the uptake and hormonal control of fluids. Intermittent hourly monitoring of blood sugar under anesthesia can help to prevent excessive hyper or hypoglycemia. In children, the rate of infusion of glucose solutions must be adjusted to maintain a normal blood glucose level throughout the operative and recovery periods.

Postoperative Care

Excellent analgesia during the operation is imperative and relative overdose must not be reversed with antagonists. It is better to support ventilation for a few hours than to reverse analgesia and subject the patient to the subsequent pain or discomfort that ensues. If the patient wakes up complaining of pain, an opioid should be titrated intravenously to obtain the required effect.

Normal cardiopulmonary monitoring should be continued during transportation and in the postoperative period. Blood replacement, if required, should continue in the postoperative period. Clotting studies indicate whether fresh frozen plasma (FFP) or platelet concentrate needs to be administered. Fluid replacement should be done to ensure a urine output of at least 0.5 ml/kg/hour.

Rewarming the patient is another priority in the recovery phase. This decreases shivering and thus, oxygen consumption; it also reverses the anticoagulant effect of hypothermia. Pethidine is sometimes beneficial to treat immediate post-operative shivering.[11] Enteral nutrition should be started as soon as possible, usually via a nasogastric tube.

Oxygen administration is always indicated in the recovery period and should be guided by ABG analysis and saturation.

Reconstructive Phase

By the time, patient reaches this phase, skin coverage has been complete and all the burn wounds have healed. Caloric requirements are supplied in excess of those needed and the anabolism has begun. The pulse rate, respiration and thermoregulation have stabilized. The anesthesia required is usually remarkably straightforward unless the face, neck or airway is compromised in any way.

Reconstruction of shape and function involve release of contractures and skin grafting, use of flaps, tissue expanders, etc. The major anesthetic problems during this phase are airway management, placement of intravenous catheters and psychological disorder, including post-traumatic stress disorder or depression. These patients may return to the operation theater for many years, so the problems tend to recur. It is important for the members of the burn team to support them psychologically and plan the surgery well ahead. Adequate reassurance and premedication are essential.

Contractures may limit mouth opening and neck movements in the patient undergoing reconstructive surgery (Figs 16.9 and 16.10A to E). Visual assessment and examination of the patient may aid in the diagnosis of a difficult airway. If the epiglottis and posterior larynx can be seen in a seated patient with neck extended, mouth wide open and tongue fully protruded, one may predict that the trachea can be successfully intubated. Neck mobility can be evaluated by asking the patient (in the sitting position) to place the chin on the chest and look downwards and then bring the head and neck all the way to look upwards towards the ceiling behind the head. Restricted neck mobility can make the intubation difficult. Another useful predictor is the "3-3-2" rule. The first '3' in the rule refers to the ability to place three fingers in the patient's mouth. This examines the adequacy of mouth opening. The second '3' refers to the space from the mentum to the hyoid bone. Three fingers placed side by side should fit into this space. This indicates adequate mandibular dimensions to permit access to the airway. The '2' in the rule requires that two fingers be placed between the thyroid notch and the floor of the mouth (i.e. the hyoid bone). This indicates that the larynx is sufficiently low within the neck to permit access by the oral route.

Contracture release on the neck may require special skills from the anesthetist as the airway may be particularly difficult to intubate. At times, the surgeon may be required to release them under local anesthesia, facilitating the intubation (Fig. 16.9). Fiberoptic intubation is the best solution for intubating such difficult airways, provided the availability of equipment and expertise (Fig. 16.10). Many times, tissue expanders are placed under normal skin adjacent to the scarred area. The bag is slowly inflated with saline solution over a period of weeks until the skin has been expanded to the desired size (Fig. 16.11). At times, the inflated expander may cause problems for the unwary anesthetist. An expander in the occipital area may cause difficulties with the extension of the neck in preparation for intubation. Similarly, an expander in the neck has the potential to cause tracheal deviation or compression. Provided that the problem is recognized, the surgeon can be asked to deflate the expander immediately before the induction of anesthesia. Other methods of intubation, as mentioned earlier, should be kept in mind and strategy planned accordingly. The laryngeal mask airway (LMA) during this phase is safe, has proved its worth and is an acceptable alternative method to try, whenever indicated.

Suxamethonium is contraindicated during this phase. Non-depolarizing neuromuscular blocking agents (NMBAs) can be safely used. In order to lessen blood loss and operate in a bloodless field, it is common to use dilute solutions of adrenaline. The anesthetic agents selected must be compatible with the surgical use of adrenaline.

Sometimes, skin grafting or local pedicle flaps inappropriate or unavailable respectively for recons surgery and the surgeon decides to use a 'free' fla operations require intricate microvascular surgery a be lengthy. Blood loss is slow but persistent. It is important that the patient's circulation be maintain optimum condition to ensure that blood flows throughout the newly anastomosed blood vessels. A of hemodilution is desirable, but the circulatory blood must be meticulously maintained and vasocons avoided.[12] The anaesthetist can make useful contrib survival of the flap. The patient must be kep throughout the procedure. Cardiovascular status sh carefully monitored. All blood loss should be repla crystalloid or colloid rather than blood should be used to achieve an optimum hemoglobin concentration of dl. A hematocrit of 30-35% will provide adequate transport, while minimizing viscosity. Tissue perfus be further improved by the administration of dex and vasodilators, which act directly on the smooth of the blood vessels in both the flap and surro tissues.[13] After surgery, it is essential to provide a analgesia, because pain causes vasoconstriction and flap perfusion. In addition, surgeon may intraoperative anticoagulation in the form of hepari

For the operations during this phase, f postoperatively is usually not a problem although a awakening is important. Coughing, restlessness and s are to be avoided, as bleeding may occur under graft may be dislodged by movement. The goal sho sedated but arousable patient.

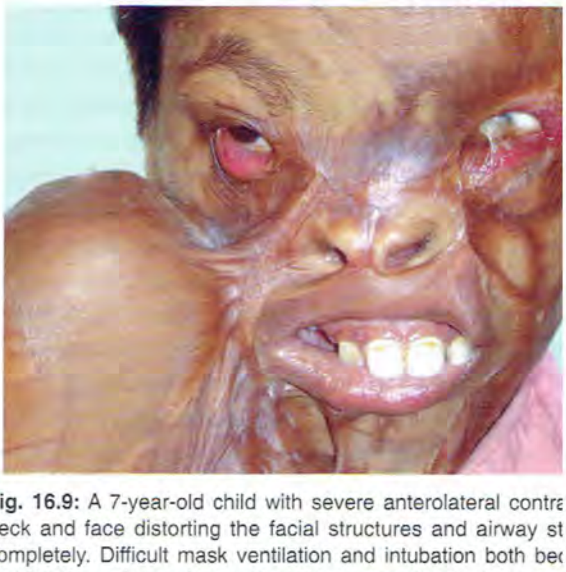

Fig. 16.9: A 7-year-old child with severe anterolateral contr neck and face distorting the facial structures and airway st completely. Difficult mask ventilation and intubation both be restricted mouth opening and inability to extend neck due to co

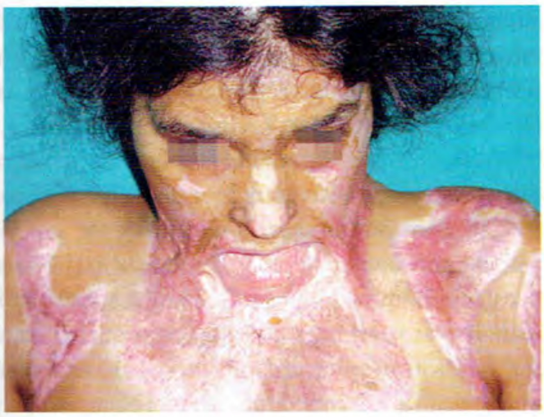

Fig. 16.10A: Severe post burn contracture neck—Front view, showing the chin touching the chest and severe flexion of neck

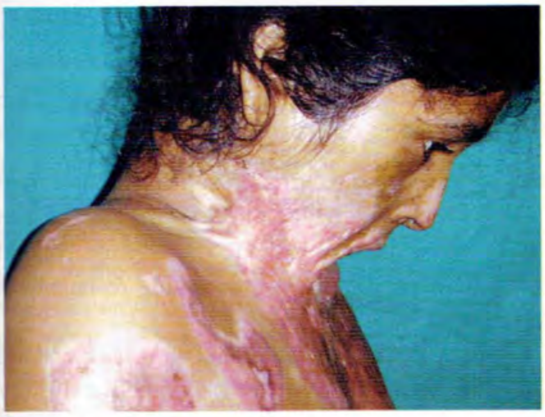

Fig. 16.10B: Lateral view of same patient showing total loss of cervicomental angle with restricted mouth opening

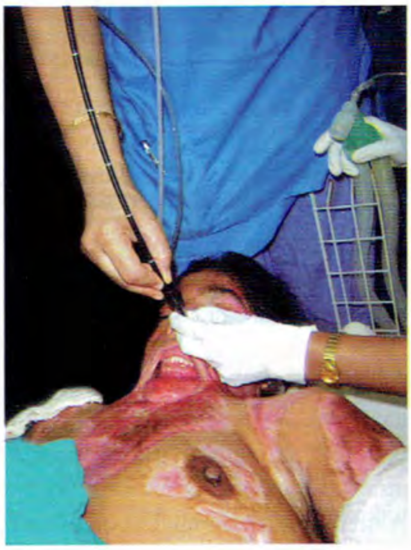

Fig. 16.10C: Fiberoptic intubation being tried for the same patient through nose

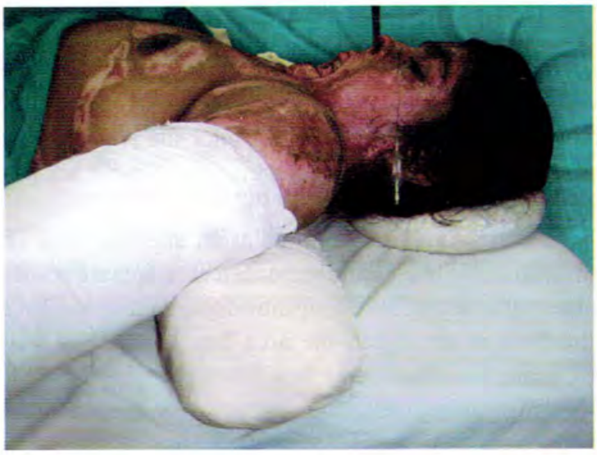

Fig. 16.10D: Patient successfully intubated

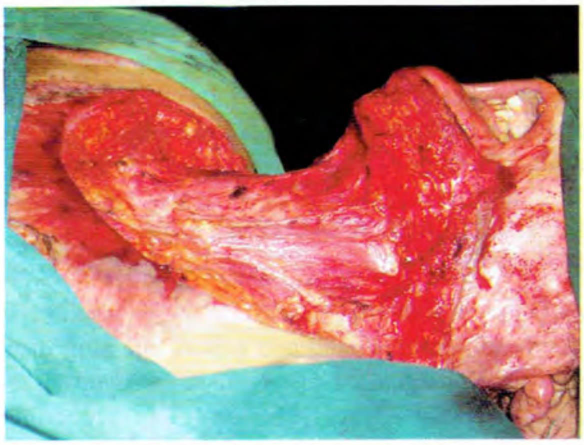

Fig. 16.10E: Same patient as in Figure 16.10D after release of the contracture showing restoration of the cervicomental angle. Neck can now be extended for easy intubation for future surgeries

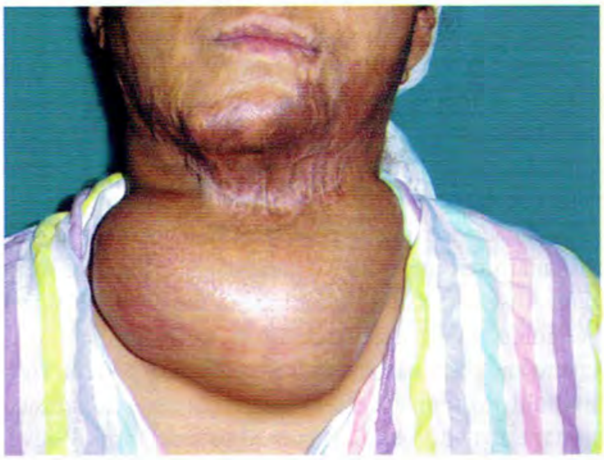

Fig. 16.11: Patient with scar on lower face with tissue expander in neck–Can create problems during intubation

LOCAL AND REGIONAL ANESTHESIA

Infiltration of donor sites with local anesthetic agents with a vasoconstrictor reduces both the blood loss and the pain produced by these wounds. If the donor site is small, topical application of EMLA cream is highly effective if anesthesia is not indicated.

The burn wound is classified as an infected area. Thus, peripheral nerve blocks and regional techniques are relatively contraindicated as the risk of introducing infection is high. If the burn is small and the area limited such as a hand burn, a brachial block may be contemplated.

During the reconstructive phase when these patients are no longer considered as potentially infected cases, local and regional techniques may be used. The sympathetic blockade produces improve blood flow to flaps by dilating vessels, facilitating tissue transfer. Probably the greatest limitation to the use of regional technique is the extent of the surgical field. Most patients with major burns have multiple operative sites along with the need to harvest skin graft/flaps from areas too extensive to be easily blocked by a regional technique.

ANESTHESIA AND ANALGESIA FOR BURN DRESSINGS

The frequency with which dressings are changed depends on the condition of wound and type of dressing material used. A severely burned patient requires many dressing changes during his/her recovery. The procedure is frequently painful and if the pain is not relieved adequately, the patient may become psychologically disturbed whenever a dressing change is due. It is also stressful for the nurses and doctors who must inflict pain to the patient. At the same time, excessive sedation following anesthesia is neither necessary nor desirable as oral nutrition gets compromised.

Prior to general anesthesia, a period of starvation is necessary, which makes the administration of an adequate calorie intake extremely difficult. It has also been said that the need for starvation makes the routine use of general anesthesia impractical.[14] It is better to have an organized anesthesia service as a part of burn management team, for the regular dressing of these patients. The benefits of such organized service certainly outweigh the disadvantage.

Opioids alone, in general, are often insufficient as pain is severe and response varies from patient to patient. Hence, individual titration to effect and frequent reassurance is important. In addition, most burned patients rapidly develop tolerance to opioids.

Ketamine is a popular choice with a maximum of 4 l
of fasting before the dressing change. Ketami
administrated in sub-anesthetic doses of 0.5 to 1 m
body weight, together with midazolam (0.5 mg/kg
weight). At these doses, the patient is not deeply uncons
and may even talk and is pain free. Recovery is us
complete in 20 to 30 minutes. If necessary, the initial
of ketamine can be supplemented with 20 mg bolus
approximately 10 minutes intervals. The duration of sed
and a number of disturbing psychological sequelae, how
at times, limits its use. Intravenous propofol and fentan
combination can also be used for dressing changes of sh
duration. The other alternatives include remifentanil inf
and general anesthesia. General anesthesia is reserve
major burns, for the very young child or for intr
dressings, e.g. to the hand, which take a considerable

SPECIAL CONSIDERATIONS

Electric Burns

The burn from electrical current results from the conve
of high voltage electrical energy to thermal en
Determinates of damage include the area of contact
duration of current flow and the varying resistance o
body.

Electrical burns can be associated with gangrene of l
and other injuries that may not be immediately obv
The extent of injury is also unpredictable. The surface in
is often small, but the extent of underlying tissue da
and necrosis is massive. Victims often have concu
injuries such as fractures of vertebrae or long b
ruptured organs, myocardial injury or numerous contus
These patients may be admitted in coma with seiz
Muscles immediately surrounding the bone are usually
affected then superficial muscles because bones ten
provide maximum resistance to the current flow and i
process produce maximum heat which causes dama
the deeper muscles. Early fasciotomy is needed to pre
the blood flow to extremities. It necessitates ge
anesthesia during the first day of injury at the time
fluid shifts, hyperkalemia and myoglobinuria are max
Massive myonecrosis and hemolysis may resu
hyperkalemia as well as myoglobinuria and hemoglobi
In the presence of hemoglobinuria or myoglobinuri
increased urine flow of atleast 2 ml/kg body weight
should be ensured by the administration of increased
and mannitol. Alkalization of urine prevents the precipit

of these proteins in the renal tubules. Follow-up of patients with electrical injuries often reveals unpredictable sequelae, which may manifest months to years later; these injures may occur in organs or areas that do not appear abnormal during the acute course of illness. These late complications frequently include neurologic dysfunction, ocular damage, damage to the gastrointestinal tract, changes in the ECG and delayed hemorrhage from large vessels. Electrical burns, most often require much more fluid administration than is suggested by the extent of surface burn because of extensive damage of deeper tissues. Therefore, use of standardized fluid algorithm is not appropriate in these cases.

Pediatric Burns

Thermal injury in children is a common occurrence, accounting for up to 40% of burn patients and majority of these are minor scalds. Children are not just small adults, especially when burnt. There are many differences to be considered.[15]

Throughout childhood, the body surface area-to-weight ratio is increased, tending towards the adult ratio the older the child gets. This results in more severe physiological responses to the burn injury. The rise in temperature may cause febrile convulsions and it may be necessary to actively cool the child. The resuscitation formulae must be adjusted to the needs of the child. Drug dosage, fluid requirements and nutritional needs are more accurately assessed if based on the surface area rather than the weight.

The inability to communicate well in the younger child present the anesthetist with a frightened, often crying child at the preoperative visit. It is important to attempt to gain the child's confidence at this time. Preoperative sedation is desirable for children to reduce the stress of fasting, transport to the operating room and induction of anesthesia. Sedation, amnesia and sufficient drying of secretions are provided with a premedicant dose of trimeprazine supported, if necessary, by a further dose of IV morphine at the time of induction.

As IV access may be difficult, an inhalation induction can be performed and a cannula placed into a vein when the child is asleep. The use of EMLA cream helps allay the fears associated with blood-sampling and intravenous cannulation. Heat loss is greater in the child because of the greater surface area to weight ratio, thin skin and higher cardiac output. Every effort must be made to maintain normal temperatures during surgery. Smaller children can be operated upon under infrared heaters. Intraoperative monitoring may prove difficult as previously mentioned, but the size of the patient adds to these difficulties. Fluid replacement, blood loss and drugs need special care as these are affected by the basal temperature, the cardiac output, the size of the burn, the size of the child and the nutritional status. The nutritional requirements are high and preoperative starvation should be kept to an absolute minimum. Hypoglycemia is common in very small children and must be guarded against by constant monitoring and an adequate dextrose infusion.

Ketamine has been used in children as the occurrence of postoperative problems associated with hallucinations is reported to be low. This may be due to inadequacy of communication skills in the child. Other techniques which includes titrated doses of various drugs like propofol, morphine, etc. as indicated are preferred that ensure minimal starvation, swift recovery and early feeding.

Blood loss, when over 10% of the estimated blood volume, should be replaced, as erythropoiesis is impaired and there is an increased need of good oxygen transport so that satisfactory healing occurs.

Pain relief should be measured objectively. Smiling faces instead of the visual analogue scale may prove successful. Children of school going age can be taught to use a patient controlled analgesic device.[16] Oral analgesics should be instituted as early as possible. Many novel ways of administering analgesia have been tried, all with some success. These include fentanyl lollipops, fentanyl skin patches and intranasal fentanyl.

SUMMARY

The anesthetic management of burn patients must include an understanding of the altered physiology and pharmacology associated with burns. Proper psychological support for the family and patient can be greatly facilitated by a thoughtful and considerate anesthesia care. The anesthetist is often the friend of such patients if he/she provides periods free of pain, including the time of transport from intensive care unit to the operation theater. It is important to remember that a normal personality resides in that devastatingly injured body[17] and we must remain sympathetic and supporting at all times and under all circumstances.

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Chapter

17

Biological Dressings and Skin Substitutes in Burns

PK Bilwani, Deepak Nanda

INTRODUCTION

The ultimate aim in the treatment of burns is to achieve wound closure. Wound closure in partial thickness burns occurs by epithelialization. The main purpose of local care with antimicrobial creams, non-stick Vaseline gauze and Gamgee pad dressings in such burns is to prevent infection and injury to the regenerating neoepithelium till epithelialization from the surviving epidermal appendages is complete. It is during this period that the burn wounds have to be covered with biological dressings and skin substitutes to reduce pain, evaporative loss of fluids, exudative loss of electrolytes and proteins and to prevent infection by preventing contamination. Once the epithelialization is complete, the biological dressings and skin substitutes either come out of their own or are peeled off. Wound closure in full thickness burns is achieved by application of autografts over the raw areas after the dead skin comes out either spontaneously or by excisional surgery. However, in extensive burns, critical level at which donor area becomes exhausted is from 30-40%; the wounds need to be covered temporarily with some biological dressing material or skin substitute till autografts are available. It is calculated that a person with 50 percent burns requires about 6,000 square centimeters of skin for coverage of the wound. Once all the burn wounds have healed, skin cover is still an issue during reconstruction.

There are various types of biological dressings and skin substitutes.[1-3] An ideal skin substitute should be inexpensive, readily available, non-antigenic, sterile, durable, flexible and permeable enough but should not allow excessive water loss. At the same time, it should provide an effective barrier to microbes. Unfortunately, such a material is not available till now. The available skin substitutes can be subdivided into two types, namely, temporary and permanent.

TEMPORARY SKIN SUBSTITUTES (BIOLOGICAL)

Amniotic Membrane

Re-epithelialization of a partial thickness skin injury is facilitated by covering the wound with an amniotic membrane.[4,5] Both human and bovine amnion has been used as a dressing material. Amnion is readily available, inexpensive and can cover large areas. Although it does not get vascularized, it provides an excellent temporary cover for a few days and enhances re-epithelization in clean second degree burns or donor areas of split skin grafts. It also enhances epithelial ingrowth from the wound margins in full thickness wounds. It exerts an angiogenic effect as well which increases capillary density of the wound bed. Amnion can be treated with 0.25 percent sodium hypochlorite and 200,000 units of penicillin, sterilized, dried and stored at room temperature up to nine months. However, its use carries the risk of transmitting diseases like AIDS. Placenta is procured from normal or cesarean section deliveries. All aseptic and antiseptic precautions are taken while handling the placenta. The placental membrane is dissected from the blood clots with a sterile gauze swab. Thereafter, it is washed with normal saline and used to cover the burn wounds. It is immaterial whether chorionic or amniotic side of the membrane is placed in contact with the wound. As it does not get vascularized, it does not get rejected by the body; thereby, allowing its repeated use.

Amnion can be stored for 2-3 days in sterile bottles containing 0.25% sodium hypochlorite solution. It relieves pain, avoids discomfort during dressing change, reduces oozing and protects underlying regenerating epithelium. It is usually advocated in developing countries where other products are expensive and out of reach. Amniotic membrane can be impregnated with antibiotics but the membrane has to be changed/replaced every two to three days.

Collagen

It is a fibrous protein of vertebrates and forms chief constituent of the fibrils of connective tissue. It can be isolated and purified in large quantities from tissues of animals. Collagen constitutes 25-30% of body protein.

Collagen is hydrophilic and very suitable for cell adhesion. It has very low antigenicity. It is commercially manufactured and is readily available in the market as preserved wet as well as dried sheets[6,7] (Figs 17.1A to 17.2). The method of application is same as for amniotic membrane. The wet collagen sheet is rinsed in normal saline and spread over cleaned wounds. Thereafter, it is left exposed to get dry and stick firmly to the wound. In case of dry collagen sheets the surface of the wound is wet with saline before application of the collagen sheet and then covered with a dressing of gauze and Gamgee. In second degree superficial dermal burns epithelization occurs under the collagen sheet. When healing is complete, the dried collagen automatically falls off. Thus, it provides good biological temporary cover in superficial and deep partial thickness burns. It also reduces pain and evaporative losses and forms a barrier for microorganisms and thus minimizing the chances of wound infection.

Fig. 17.1A: Dry collagen sheet (Skin Temp®)

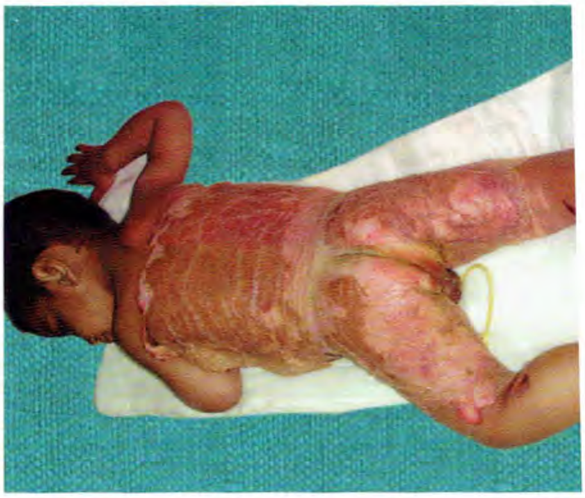


Fig. 17.1B: Dry collagen sheets applied to an extensive bu child–Majority of the wounds healed within 10 days without a of infection

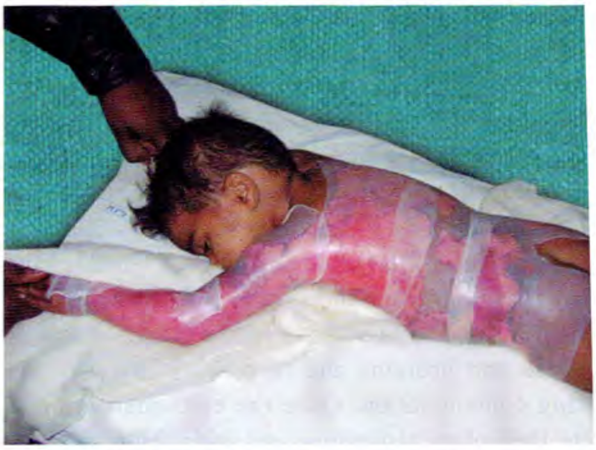


Fig. 17.2: Wet collagen (Kollagen®) applied to another case of ex burns in a child–The wounds are sealed from the environme heal fast

Like amnion, collagen does not get vascularize hence adherence to the wound is poorer as compar skin grafts.

Homograft (Human/Cadaveric Allograft)[8-11] (Figs 17.3A to 17.4C)

In 1881 Girdner treated burns with skin harvested f suicide victim. Schede used allografts same year harv skin from fresh amputation specimens and cadavers. then, many workers have used skin allografts as temp biological cover in burns. It serves as a temporary co partial thickness burns preventing evaporative lo proteins and electrolytes besides relieving pain and

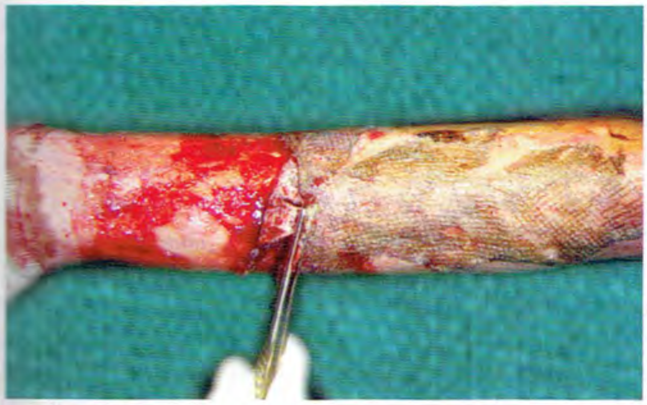


Fig. 17.3A: Homograft from related donor applied to a patient with extensive burns–On removal of the homograft it shows healthy granulating wounds

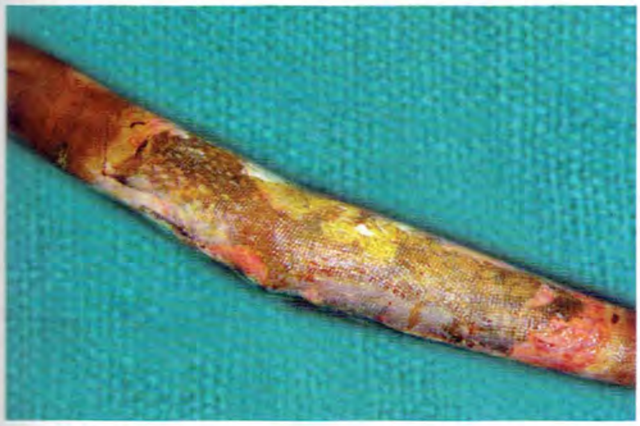


Fig. 17.3B: After removal of homograft, autograft was applied and fully 'taken' up

of dressings. Skin allografts have also been used extensively for preparing the wounds for grafting. Because of availability of large skin banks the source of homografts in western countries is cadavers. However, due to non- availability of skin banks and lack of public education about skin donations the surgeons of developing countries procure homografts from live donors – preferably near relatives. Non-availability of artificial skin mandates salvage of major burns by homografts.

The senior author (PKB) has been using skin homografts in salvage of major burns (above 40%) since 1985. In almost 94% of these cases the source had been voluntary donors since skin bank facility is not available everywhere. Indian community is the most wonderful community in the world where parents and near relatives are always willing to get admitted and undergo surgery to donate skin for their relative. In our experience, the homografts are mostly required in

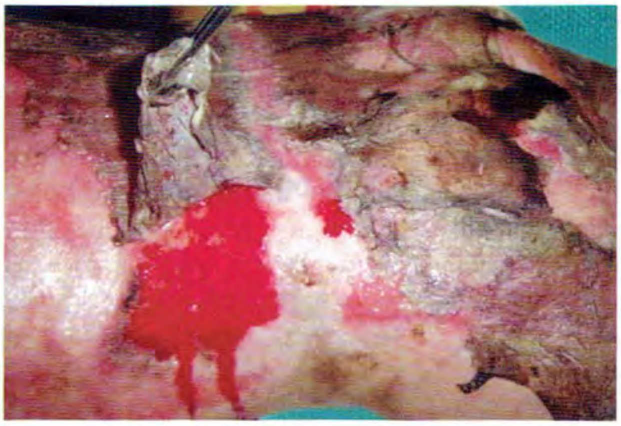


Fig. 17.4A: Homograft from mother applied in a 4-year-old with extensive burns–Being removed in multiple sittings for autograft application

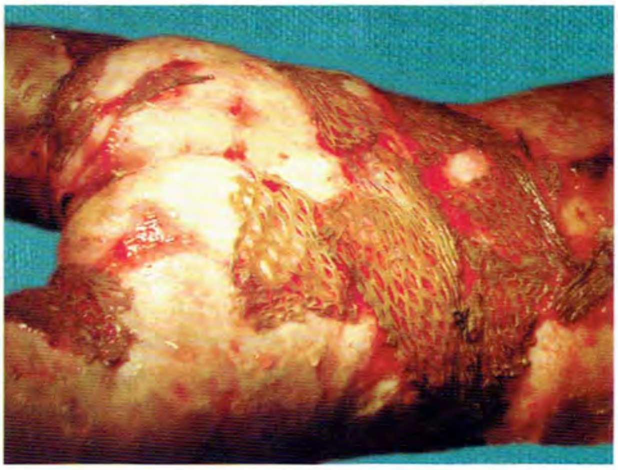


Fig. 17.4B: Autograft applied on healthy raw areas

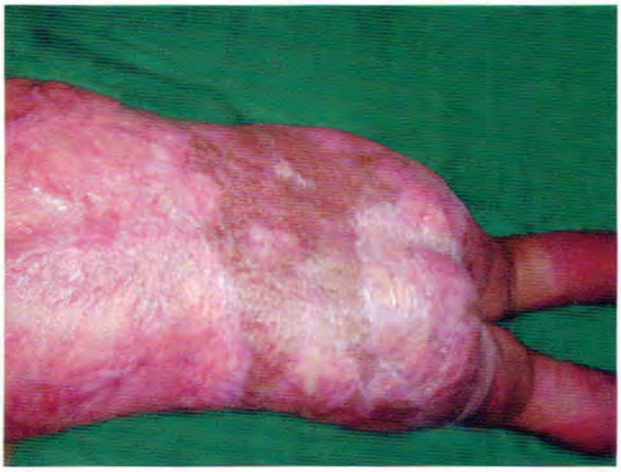


Fig. 17.4C: Completely healed areas after 1 month

patients with 50-70% burns. Early excision in this group of patients is not always possible due to obvious reasons and they are treated conservatively till all second degree burns have healed and the third degree burns start granulating. Thereafter, the important areas are covered with whatever autografts are available and the rest are covered with homografts. Once these grafts settle (10-12 days), the patient's general condition improves fast. The homografts get vascularized or 'take' exactly like an autograft. They survive for 3 to 10 weeks before getting rejected by the body's immune system. They should be removed from the wound before the signs of rejection appear as the rejection reaction can deteriorate the condition of the wounds. For the coverage of resultant raw areas, autografts can be easily reharvested.

As the homografts get vascularized like autografts (particularly the fresh homografts), they form best biological dressing material in reducing pain and the bacterial density of the wound. If skin bank facility is available the homografts can be freeze-dried and stored for longer time though their viability diminishes as the time passes.

Heterograft (Xenografts) (Figs 17.5A to C)

As homografts are difficult to procure, surgeons have used skin procured from porcine or bovine or canine sources. However, the xenografts do not get vascularized and hence, their tissue adherence property is poor and not much effective in preventing bacterial proliferation. They do prevent evaporative losses and relieve pain. The religion of the
poses a hurdle in the use of xenografts with respect
source.

TEMPORARY SKIN SUBSTITUTES (SYNTHETIC)[12,13]

The synthetic skin substitutes (synthetic dressing mat
though available "Off the shelf", lack property of ad
and hence sub-membrane suppuration is common
various synthetic skin substitutes consist of film
polyurethane (Opsite, Biobrane). Foams like polyur
foam (Looms), gels like amiderm, duoderm, Kaltost
and sprays like polyhydroxyethylene methacrylate.

Biobrane (Winthrop Pharmaceuticals, Santa Ana, CA)

It is a two layered temporary, synthetic skin substitut
outer layer is composed of ultrathin layer of silicon
which is semi-permeable and permits the exit of
vapours but prevents entry of bacteria from the ex
The inner layer is tightly woven nylon fabric. Both
have porcine collagen peptides covalently bonded
allows the sheet to adhere to the wound by integrat
tissues without causing inflammation. The collagen is
replaced with host tissue. Biobrane (Fig. 17.6) is trans
allowing observation of wound healing through it. It is
and confirms to irregular surfaces. When donor sit
autografts are available, the silicon sheet is remove

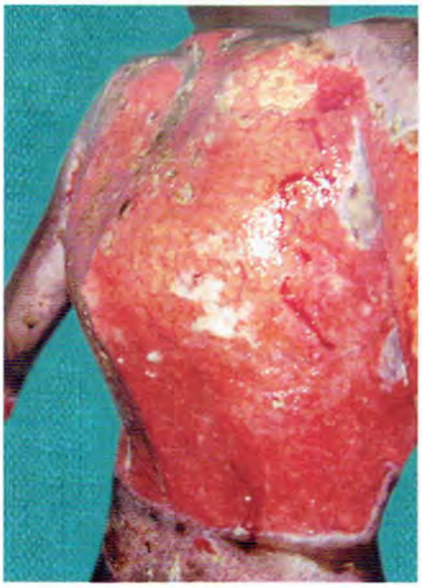


Fig. 17.5A: Granulating burn wound

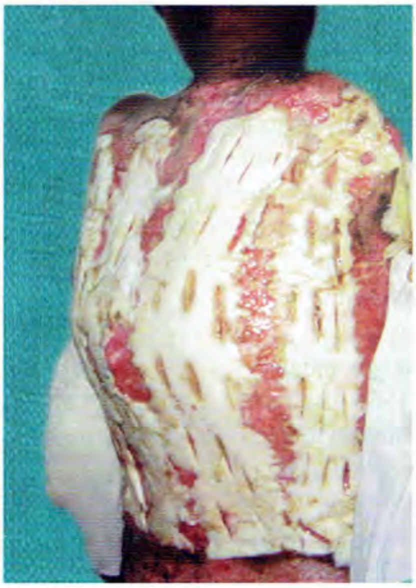


Fig. 17.5B: Wound covered with porcine xenografts

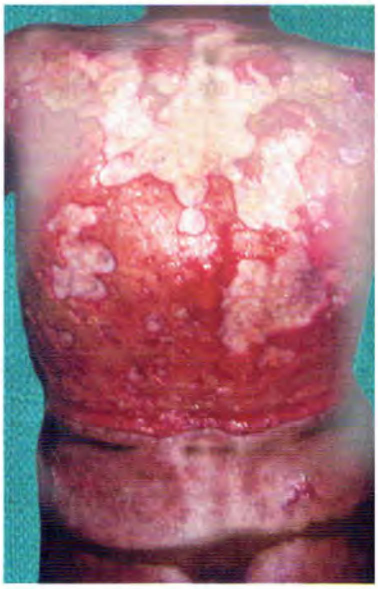


Fig. 17.5C: Epithelialization under po xenografts

autografts are applied. It should preferably be removed in about two weeks to prevent fibroblast ingrowth into the mesh.

Transcyte (Advanced Tissue Sciences, La Jolla, California)

It is a bi-laminate synthetic, temporary skin substitute. The dermal layer is composed of a layer of collagen synthesised by human neonatal fibroblasts cultured on a nylon mesh. The epidermal analogue is made up of silicone. It is better than cadaveric skin as it can be easily removed with less bleeding but leads to equal or better take of autografts. There is no apparent immune mediated rejection.

Omniderm (Omikron Scientific Ltd, Rehovat, Israel)

It is a temporary synthetic skin cover which is thin, transparent and flexible membrane of polyurethane. It is hydrophilic, absorbs water and is elastic in nature. It is permeable to liquid and can be used as an interface for skin grafting (as a layer between graft and the dressing). It allows water vapor to evaporate and allows antibiotics to permeate inside.

Apligraf (Graftskin)(Organogenesis Inc., Canton, MA)

This manufactured skin analogue is composed of two layers, viz., the epidermal layer made of human foreskin derived neonatal keratinocytes and the dermal layer made of neonatal fibroblasts seeded in Bovine Type 1 collagen lattice. This has been shown to improve the cosmetic results when applied over widely meshed grafts.

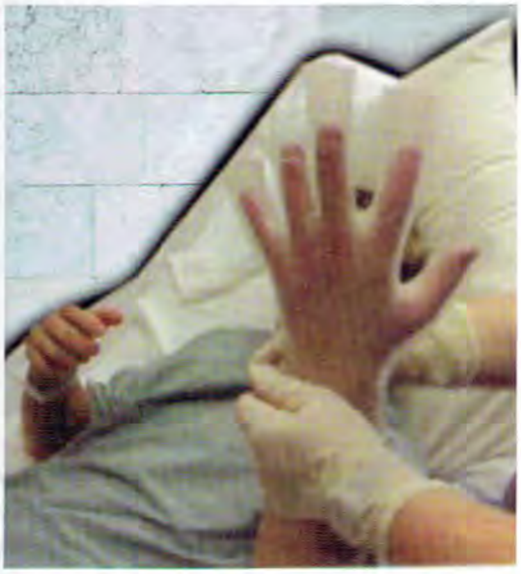


Fig. 17.6: Biobrane applied over hand burn (*Courtesy:* Smith and Nephew Ltd.)

Opsite/Cling Film/Via Film

It is a synthetic adhesive moisture vapor permeable dressing made up of modified polyvinyl chloride. Although it is permeable to water vapor, it is impermeable to bacteria. The permeability to water is low; at times can lead to collection under this dressing. Pain relief, lack of bulky dressings and early healing are the advantages.

PERMANENT SKIN SUBSTITUTES

Dermal Replacement-synthetic

Integra (Integra Life Sciences Corporation, NJ) (Figs 17.7A to G and 17.8A to D)

This is a synthetic, bi-laminate product designed to provide permanent skin cover, with outer epidermal substitute made up of silicone (polysiloxone polymer) layers which controls moisture loss from the wound bed. The deeper dermal analogue is made up of bovine tendon collagen arranged in a three dimensional porous matrix and cross-linked with glycosaminoglycan (chondroitin-6-sulfate, derived from shark cartilage). With its use the dermal layer is infiltrated by fibroblasts. This is followed by capillary invasion which vascularizes and leads to deposition of collagen by fibroblasts and the dermal matrix gradually biointegrates with the wound. The silicone layer separates out in 2-4 weeks time when a thin split skin graft coverage shall be required to cover the neo dermis.

Integra has been used since 1981(Burke et al) and subsequent trials have concluded that hypertrophic scarring was less, late contractures were rare and artificial skin grafted areas demonstrated growth with the growth of the child.

Derma Graft (Advanced Tissue Sciences, La Jolla, California)

This synthetic dermal matrix is made by seeding neonatal fibroblasts on to a polyglactin vicryl mesh. It is usually used on excised burn wounds with a split skin autograft. Gradually over a month the mesh gets absorbed. The graft uptake on the matrix is slightly less than autograft alone but shows more resistance to bacterial contamination.

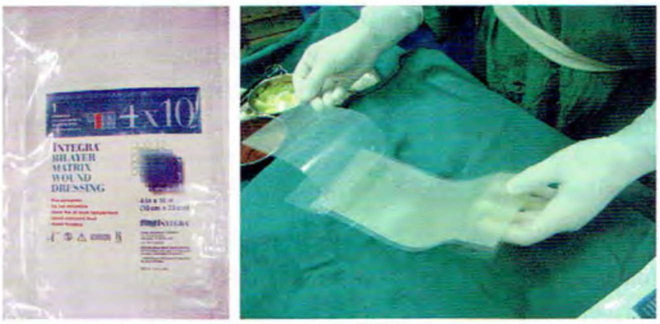


Fig. 17.7A: Sample of integra (*Courtesy*: Integra lifesciences corporation)

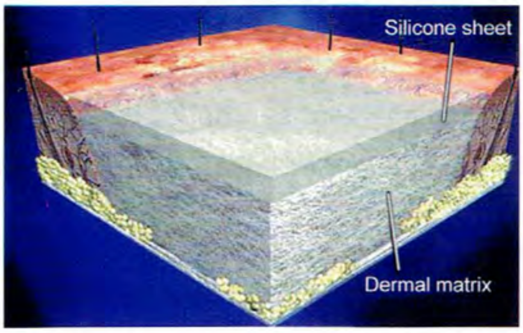


Fig. 17.7B: Line diagram of integra showing the upper silicone sheet and lower dermal matrix

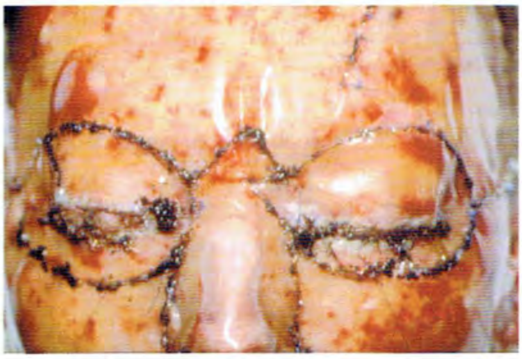


Fig. 17.7C: Integra applied on face following excision of deep burns (*Courtesy:* Integra lifesciences corporation)

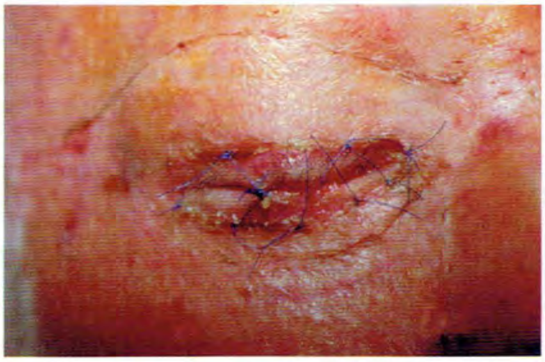


Fig. 17.7D: Following application of skin graft

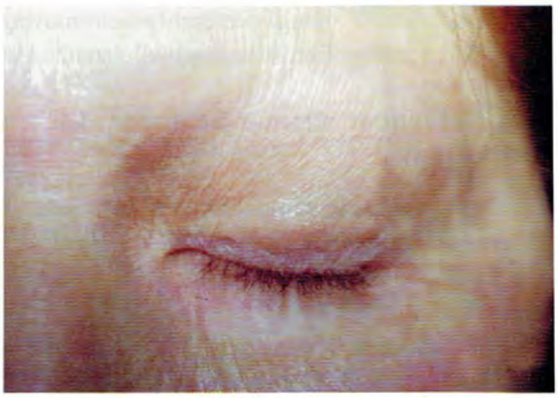
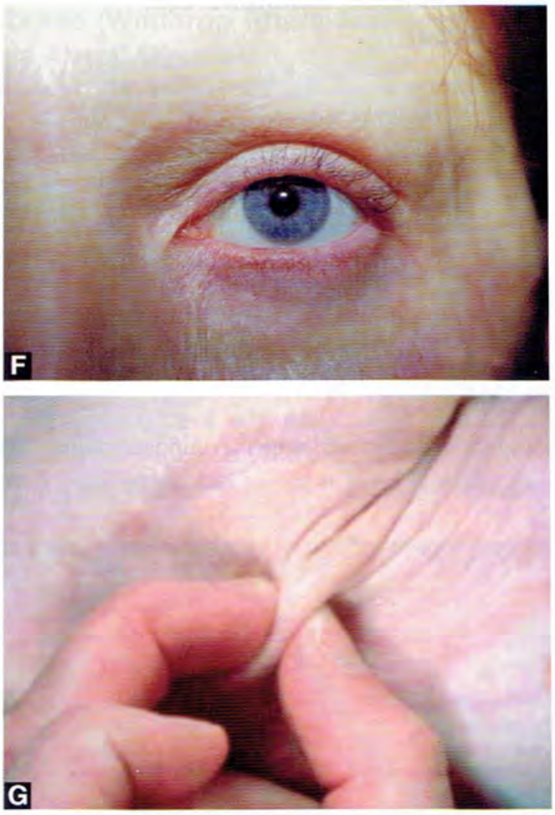


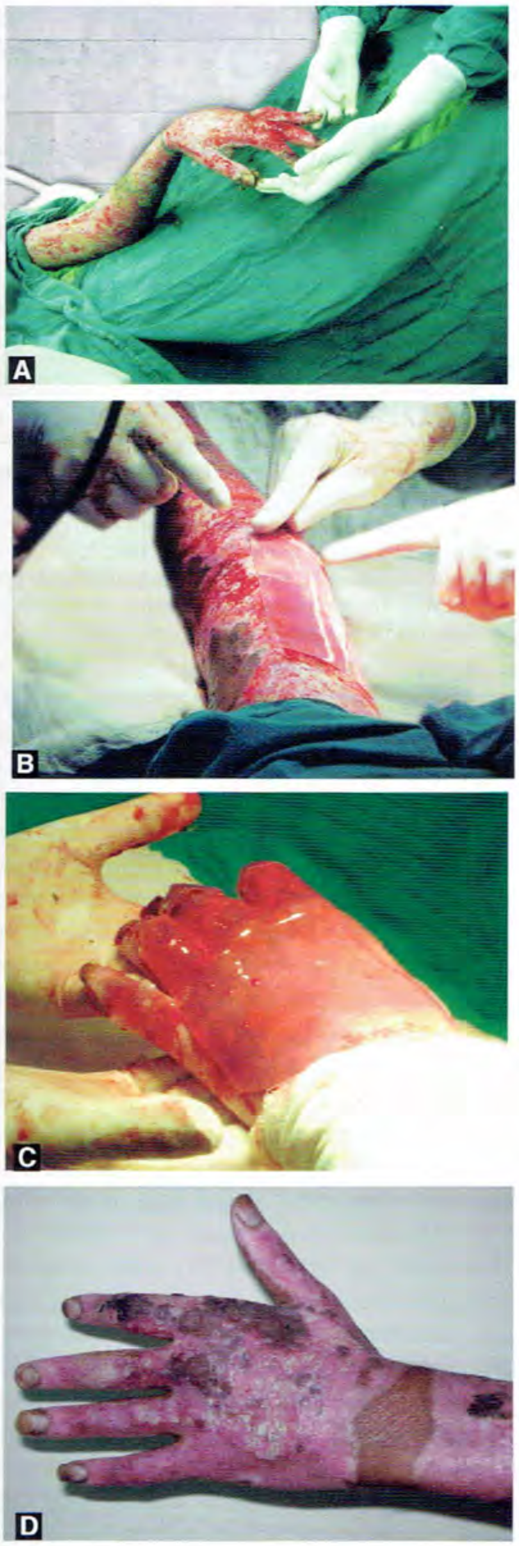
Fig. 17.7E: Quality of skin following complete healing of w



Figs 17.7F and G: Unrestricted mobility of lids

Dermal Replacement—Biological

Alloderm: It is de-cellularized allograft dermis. It is pre
by a special biochemical process to make the human
acellular. It is used as a dermal transplant in deep s
degree and third degree burns. Donors are screened fo
1 and 2, HBV, HCV, HTLV 1 and Syphilis serologically
the material is freeze dried and stored. After wound ex
the dermal matrix is grafted on to the fresh woun



Figs 17.8A to D: (A to C)Application of Integra after excision of deep dermal wounds on upper arm and dorsum of hand. (D) Final healed area on the dorsum of hand after removal of silastic sheet after 21 days and application of thin split skin graft

covered with split skin graft. There are fewer incidences of inflammation and infection.

EPIDERMAL REPLACEMENT

Cultured Keratinocytes (Epidermal cells)[14,15]

Rheinwald and Green in 1975 found that irradiated murine fibroblasts can support the growth of human fibroblasts. In this technique a culture medium of fetal calf serum is used to grow keratinocytes into confluent sheets of epidermis. These epidermal sheets are usually two to eight layers thick. By regular sub-culturing, a three square centimeter sheet can be expanded five thousand times yielding almost enough epithelium to cover the whole body surface of an adult human. This level of expansion requires sophisticated labs and a large number of staff and is routinely impracticable. This topic is covered in more detail elsewhere in the book.

Cultured Allogenic Keratinocytes

This has the advantage of unlimited supply and is immediately available. Cultured epidermal allografts have been successfully used even after cryo-preservation. Initially the survival of this allograft was thought to be because of elimination of Langerhans' cells during the cultivation process which is assessed by a lack of HLA-DR expression. Indefinite cell survival in the allograft is controversial, the long term wound healing is suggested to be due to the residual host keratinocytes whose growth and migration is stimulated by cultured allografts. The disadvantage is that it still carries the possibility of disease transmission.

COMPOSITE EPIDERMAL AND DERMAL REPLACEMENT

Composite (Dermal-epidermal) Cultured Grafts

Although cultured epidermal sheets have been successful in some cases, they have following disadvantages (1) Epidermal sheets lack dermis and therefore do not vascularize adequately on full thickness wound beds and hence do not adhere satisfactorily. (2) Epidermal sheets lead to extensive scar formation. (3) In normal skin presence of rete ridges at dermal epidermal junctions (DEJ) provides resistance against shearing forces. However, following application of cultured epidermal sheets in full thickness wounds, the epithelium is unstable due to the lack of rete-ridges and may get easily detached.[5]

The above disadvantages with the use of cultured epidermal sheets clearly indicate that dermis component is essential for satisfactory wound healing. Hence, for optimal wound healing Dermal-epidermal grafts are developed by growing epithelium on a dermal substrate. The dermal substrate is derived from either collagen-gel-fibroblast or Allograft Dermis.

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Chapter 18

Skin Banking

Sujata Sarabahi, Deepak Nanda

INTRODUCTION

An extensive burn injury involves loss of large amounts of normal skin leaving insufficient donor sites for coverage of wounds with skin autografts. The best treatment for a partial thickness burn wound is its coverage with some dressing which can protect it from infection and injury and allow it to heal at the earliest. Full thickness burns, obviously, need coverage with skin graft. Skin is the best biological dressing whether it is autograft, homograft or xenograft. Homografts have been in use for treatment of extensive burns for many decades as they provide an easy and practical method for temporary wound closure, despite having their own limitations. Homografts can be obtained from either living donors or cadavers. It is usually difficult to get fresh homografts in large amounts unless the donor is a live-related donor which is a very rare situation. For such cases the grafts may be harvested as and when required and therefore, banking of the skin is not required. Cadaveric skin grafts are a good choice when requirement is large. Their use in extensive burns requires establishment of skin banks for their storage till such time that they can be used. This is because they have to be harvested even when there is no immediate requirement of the homograft.

Skin banks provide facility to store not only cadaveric skin but also for the skin from amputated parts, xenografts and amnion, etc. With the establishment of skin banks in association with major burn centers, the morbidity of extensive burn patients has reduced dramatically.

It is well known that the viability of skin graft begins to decline as soon as it is harvested and lifted off the donor site. The rate of decline depends on many factors, the main being the temperature at which it is stored. The cellular viability is inversely proportional to the temperature at which it is kept. If the skin grafts can be rapidly cooled in a well oxygenated medium, their viability may be prolonged.

Wentscher[1](1903) was the first to refrigerate free skin as autografts, one sample for seven days and three for fourteen days. All but one of them survived. After continuous experiments, Carrel[2, 3] concluded that Vaseline was the most satisfactory medium for refrigeration and in 1911 he suggested to the surgeons that refrigerated skin can be used for clinical advantage. Kubanyi (1930), in his experiments, preserved autografts in patients own serum and applied it after 12 days of their harvest (as quoted).[4] He showed that these grafts survived for 8 days after transplantation. It was, therefore, established that apart from providing a low temperature, a cryo-protective medium is also essential to maintain the cellular structure of the graft.

METHODS FOR SKIN STORAGE

There are various methods for storing the skin graft.

1. **Refrigeration:** It is the most commonly practiced method as it is easily available at every centre. The graft is kept in the refrigerator at 4°C in a sterile bottle after wrapping it in a Vaseline gauze (epidermal side on gauze) followed by saline soaked gauze pieces. The raw surface of the graft is to be kept in contact to prevent excessive evaporation. When grafts are required for clinical use, the outer layers of Vaseline and saline gauze are removed and grafts washed with saline before being applied to the raw areas in the same manner as fresh grafts.

   This method is used mainly for autografts and in this manner the grafts can be stored for up to 3 weeks although the viability of the graft decreases with time. It is best used within a week after which there is a definite deterioration in the skin structure affecting its 'take'. This hypothermic storage technique can be improved to prolong the useful period of graft storage by keeping the grafts in a cryo-protective medium like dilute homologous serum, tissue culture medium or balanced salt solution as sources of nutrient to the skin cells.

2. **Deep freezing or cryo-preservation**: It involves the cooling of tissues to ultra low temperatures. However, at below 0°C temperature, there is disruption of cytoplasmic contents of the cells leading to a mechanical rupture of the cells. To reduce this effect and improve the cell viability, the tissues have to be pretreated with cryo-protective agents. They can now be slowly cooled to very low temperatures with minimal molecular damage. The agents used are glycerol, ethylene glycol, dimethyl sulfoxide, etc. whose freezing points are below minus 300°C so that they do not form any crystals. The cooling is done by using liquid nitrogen to temperatures as low as minus 80 to minus 196°C and then stored by placing them in deep freezers or submerging them in liquid nitrogen. Usually liquid nitrogen flasks are used for transporting homografts from skin banks to the burn centers for use. Grafts cooled to minus 80°C can be stored for 6 months while those cooled to minus196°C can be stored indefinitely.
3. **Freeze drying or Lyophilization:** It is the process where the graft is first rapidly cooled and then the water is removed by sublimation and the tissue is vacuum sealed. These grafts are sterilized by gamma irradiation. These freeze dried grafts are stored at room temperature and are nonviable since the skin cells do not survive dehydration. These grafts do not 'take' and serve as a biological dressing only.

HOMOGRAFT SKIN BANKING

Harvesting

Split skin grafts are harvested from refrigerated cadavers within twenty four hours of death. Signed consents are required from appropriate relative(s) for the purpose. Skin donations are acceptable from individuals between the age of twelve and sixty years with no history of malignancy, hepatitis, jaundice, dermatological disorders and venereal diseases. Blood samples taken from the cadaver are tested serologically for AIDS (HIV 1 and HIV 2), Hepatitis B, Hepatitis C and syphilis.

All the donor sites are shaved and prepared. Skin are harvested taking all aseptic and antiseptic me exactly similar to surgical operations being carried live patients. Using dermatomes strips of split skin are harvested in thickness varying between 0.25 n 0.35 mm from limbs and trunks. After harvesti representative piece from each area is sen microbiological testing for *Streptococcus*, *Pseudomon* *Staphylococcus*. Any skin graft whose sample pro more than 10 organisms/gm of donor tissue, is disc as it is unsuitable for treatment of burn wounds.

Cryo-preservation[5]

After the skin grafts have been harvested they are s on meshed Vaseline gauze with epidermal side on the g The length of skin strip dictates the number of time skin has to be folded on itself. It has been seen that stri skin 40 cm or more in length have to be folded twice. sheets are then rolled and packed into sterile pl containers. These containers are immediately immers a sterile solution of 15% Glycerol and Ringer Lact 4°C for two to four hours. This period provides tim the cryo-protectant to enter the cells of the tissue in to prepare the skin for freezing later. Other cryo-protec which are used are Dimethylsulphoxide, Ethane-diol Propane-diol but Glycerol is still the most commonly In DMSO the time required for the cryoprotectant to cells is 30 min. whereas in glycerol it takes a minimum hours. After about four hours the solution is poured of the skin strips are transferred to polyester plastic envel which are heat sealable. The envelopes are then lab properly with name, mortuary number of the patient the date of skin harvest and thermo sealed. The envel are weighed and the size of skin and the weight are reco

If the harvested skin is urgently required, the gly solution step is omitted and skin stored in the refrigerat 4°C in sealed envelopes as above. In this way, the skin be stored for a maximum of 10 days. However, it is used within 3 days by which time serological test resul AIDS and Hepatitis, etc. are available.

Freezing of skin can be done in two ways, controll uncontrolled. Controlled freezing involves bringing temperature down by 1° per minute to about minus ei to minus hundred degrees Celsius while in uncontr freezing the skin strips are directly transferred to refrigerator at minus 70°C or into liquid nitrogen v where temperature goes down to about minus 100° controlled cooling, the viability of skin keratinocytes fibroblasts is maintained but the immunogenicity is reduced (as assessed by mixed epidermal cell/lymphocyte response) by 95% in murine allografts. The major immunostimulatory cells in the skin are the Langerhans' cells which are differentially inactivated. Temperature probes are embedded into the deepest layers of skin to give a feedback and from these it is evident that controlled cooling is better than the uncontrolled one. The latter produces ice crystals inside the cells which cause mechanical damage and it also changes intracellular osmolality (produces hyperosmolality) which affects skin viability. This is more evident in thin flat packets which undergo rapid cooling. After controlled cooling, the skin packets are stored either by being submerged in liquid nitrogen or in electrical freezers with modified aluminum drawers having slots for skin packets. Skin in this condition can be stored indefinitely till it is required. The disadvantages of storage in liquid nitrogen are that both loading and retrieving are cumbersome. Proper ventilation is required in the room that houses the liquid nitrogen apparatus.

Following the harvesting of a skin graft, the vascular supply of the cells is totally cut off and there is a decline in skin viability because of the following reasons:

1. Lack of nutrients and oxygen.
2. Build up of toxic materials.
3. Generation of free radicals causing lipid peroxidation
4. Osmotic changes
5. Uncoupling of biological pathways.

Addition of homologous serum in a concentration of 10-35% has been found to be beneficial in reducing this decline in the tissue viability. Not only it provides nutrition to the graft but also dilutes and buffers the acids produced during metabolism. Tissue culture medium is also used to provide nutrition and neutralizing harmful metabolic by products.

The requirement of oxygen has been found to be temperature dependent. At 6-8°C cells require oxygen but at 0 degree and below there is no requirement of oxygen any more and in fact, it is detrimental. Hyperbaric oxygen and 100% oxygen have been found to be harmful and this probably indicates the detrimental role of oxygen free radicals. Enzyme Superoxide Dismutase helps in maintaining viability of stored skin, the higher the level of SOD the longer the period of cell survival.

Osmotic changes take place in stored cells which are detrimental to the cells. These changes are due to slowing down of Na-K-ATPase pump. At 5°C, it works at about 1% of its normal capacity with subsequent influx of sodium, chloride and water leading to cell edema. This can be prevented by adding impermeable solutes to make the solution hyperosmotic or by using an intracellular salt solution to obviate the effects of ionic differences between intra and extracellular compartments. University of Wisconsin solution and CP Test are examples.

Although our understanding of detrimental effects of low temperature on tissues and cells has improved, not much has been achieved in improving the process of hypothermic skin storage.

Rewarming

When the stored skin graft is required, a rapid re-warming has to be done so that its viability is preserved. It is done by re-warming at a rate of 50-70°C per minute. This can be done either by placing the packets in a microwave oven at lowest microwave setting or by placing the packets into a two liter jar of water at 42°C until there is equilibrium between skin and water. Water gives rapid warming irrespective of size of packets. Skin stored in flat packets can be warmed ten times faster than the skin rolled into cylinders. The longer time taken to thaw the skin stored in cylinders is disadvantageous for the skin. The other disadvantage is that the cylinders occupy more space. The skin graft, after being thawed, has to be applied within two hours.

Some experiments have been done to test the viability of stored skin including enzyme levels of cells or oxygen consumption by the skin. Wachtel et al[6] conducted experiments comparing the viability of fresh allograft, frozen allograft thawed in a microwave, frozen allograft thawed by saline bath and fresh autografts. All the grafts were applied as a mesh. He found that in autografts, the graft adhered and the interstices closed in twelve days, fresh allografts adhered but interstices closed in sixteen days, the frozen allografts whether thawed by microwave or saline-bath adhered for about thirty days but did not grow. Histological examination shows that in 3-weeks time most of the epithelium dies. The dermis is edematous with chronic inflammatory cells and there are no signs of neovascularization. Both the skin samples, whether thawed by microwave or warm saline, showed no histological differences. They also found that samples with more than 10 organisms were involved with rejection. Refreezing and thawing decreased viability by about 40 percent.

Microbial assessment of the skin is done, while in storage, as this skin has to be applied to immunocompromised burnt patients. Bacterial enzymes and toxins reduce tissue viability. Testing is done for *Staphylococcus epidermidis*, *Staphylococcus aureus*, *E.coli* and *Asperigillus*. May and Wainwright[7] found that one skin sample yielded correct skin microbial assessment 92% of times, five samples yielded correct microbial assessment in 96% of times and ten samples yielded 99.9% correct results.

Pre harvesting preparation of skin is the most important factor in controlling infection of the stored graft. Protection against AIDS and Hepatitis B and Hepatitis C is vital and serology must be done carefully and routinely for donors. For medico-legal purposes, it is advisable to have serological tests done on the recipient patient as well.

FUTURE

Continuous efforts have been made over the years to improve the chances of 'take' of homografts. Cryo-preserved homograft were abraded to remove the epidermis and then super-imposed with cultured keratinocytes as described by Cuono et al.[8] Removal of homograft epidermis eliminates most of the HLA-DR antigens. GaoZhi-Ren et al[9] from China inoculated homograft skin with uncultured epithelial cells into the dermal layer while still in situ on the cadaver. The combination was removed and grafted to the patients. The epithelial cells grew by day forty and covered the whole wound.

Sawada[10] from Japan used small pieces of autografts (3 × 3 mm fragments 10 mm apart) and covered with big sheets of meshed homograft skin. Yang et al[11] described something similar where they made holes in the covering homograft skin and introduced small pieces of autografts.

Homograft 'take' is helped considerably by immunosuppression, like other organs, as shown by Burke et al.[12] They used Azothioprine and anti-thymocyte globulin which are highly toxic. Cyclosporin A has gained a lot of popularity due to its efficacy and safety. Achauer et al[13]showed that when Cyclosporin A was given for 120 days homograft skin stayed viable for two years.

SUMMARY

Cadaveric homografts have an established role to play in saving an extensively burnt patient. For them to be most effective, they must be properly harvested, microbiol tested and correctly frozen and stored. Though the skin homografts has its own limitations becaus immunogenic response, techniques are being devel overcome these problems in future. The skin allogr inexpensive compared to other organ transplants ex process of banking which is costly and therefo available at all burn centers. There is, therefore, a establish more of such skin banks so that the fac cadaveric homografts is available for treating extensi patients at all centers.

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Chapter

19

Keratinocyte Culture

RP Narayan

INTRODUCTION

An early burn wound healing is a cherished desire not only of the burn victim but also of the treating burn surgeon. Superficial dermal burn wounds heal spontaneously within three weeks if optimal conditions for healing are ensured. Very deep dermal burns either take very long time (up to 6 weeks) or may even fail to heal with lots of undesirable complications like contracture and ugly scars, etc. To circumvent these, surgical intervention in the form of skin grafting is required. Finding requisite amount of split skin graft from available donor sites is a common problem in extensively burnt patients. A large variety of skin substitutes and dressings are used to temporarily solve the problem.

The management of burn wound has evolved over the past several decades. Early excision and grafting has become the standard of care for surgical management of the burn wound. In cases of extensive burns where total body surface area burnt may exceed the available donor sites, there arises a need for replacement of human skin. The skin replacement technologies introduced over the past two decades or so have positively impacted burn care. Efforts have been focused on temporary and ideally permanent replacement to native human skin.

Serious short and long term problems occur when the epidermal and dermal layers of skin are destroyed. The short term problems, which can be life threatening, include the loss of protein rich fluid and invasion of bacteria and other microbes. These essential barrier functions are provided by the epidermis which consists of keratinocytes attached to an underlying basement membrane. The dermal layer provides for the durable and pliable characteristics of skin, which are so vital to proper skin function and cosmesis. When available, the replacement material of choice is the patient's own (autologous) skin to treat acute burns. Autologous split-thickness skin graft is closest to being the ideal skin substitute. One of the major problems in extensive burns is the relative lack of available replacement by autologous skin and the well known limitations of homograft. This brings to attention the importance of skin culture and how it can help us overcome the above problems.

HISTORY OF KERATINOCYTE CULTURE

Healing of partial thickness burn wounds (epithelialization) occurs from basal epithelial cells (keratinocytes) of epidermis and skin appendages (hair follicles and sweat glands). Attempts of growing autologous keratinocytes in laboratory for coverage of burn wounds have been in practice for the last few decades. Keratinocytes have been known to be difficult cells to grow because of their limited proliferation, early differentiation and frequent contamination with fibroblasts. Cultured epidermis for human use in burns was first proposed by Pickerill in 1951[1] and in 1956. Basset, Evans and Earle[2] also proposed that epidermal cell culture in massive quantity could provide a solution for wound closure in burns. Since then, efforts have been made to develop techniques for growing the transplantable epidermis (Karaseck, 1968)[3] *in vitro*. The importance of fibroblasts in epidermal growth and differentiation has also been demonstrated. Later on, works by Green and associates (Rheinwald and Green, 1975[4]; Green et al 1979[5]) have demonstrated the techniques for serial cultivation and differentiation of keratinocytes and proposed logistical estimates for human application. The first clinical experience with cultured autologus epithelium was reported by O'Connor et al [6] in 1981 and later by many other teams from all over the world (Gallico 1985[7]; Cuono, 1986[8]; DeLuca 1992[9]). The biggest advantage of this technique is the enormous expansion potential of a small skin biopsy into a live cultured epithelium ready for grafting large burn area in three weeks (Paddle-Ledinek et al, 1997).[10] Besides this, cultured epidermis has a wider application spectrum as in the treatment for skin ulcers (Hefton et al 1986)[11], vitiligo patches (Plott et al, Lontz et al, 1994)[12,13]and other skin defects (Green, 1991; Hafemann et al, 1994).[14,15] Human epidermal keratinocytes have been shown to grow from small biopsy samples of a patient by use of co-culture methods. They proliferate and form thin, multilayered epidermal sheets that have been used to resurface severely burned tissues. After transplantation, these epidermal sheets or cultured epithelial autografts form an organized epidermis and reestablish epidermal coverage, with formation of a new connective tissue or a neodermis beneath the new epidermis.

TYPES OF KERATINOCYTE CULTURE

Autologous Keratinocyte Culture

It is a highly technical and skilled procedure requiring a sophisticated tissue culture laboratory. Briefly, it has the following components:

Patient Selection

Patients with extensive burns, generally taken as more than 50% total body surface area (TBSA) burnt with majority being third-degree, in whom the raw areas exceed the available donor sites, are the ones suitable for this procedure. Patients having cutaneous tuberculosis, AIDS or Hepatitis B are not suitable candidates for culture. Age is no contraindication. However, an elderly with a major medical problem is a definite contraindication as they compromise the prognosis. Inhalational injury is also not a contraindication.

Biopsy

There should be a minimum delay between the burn injury and the plating of keratinocytes. Preferably it should be less than 48 hours. If it is delayed beyond 48 hours there is usually a bacterial contamination of the skin requiring antibiotic treatment of the biopsy *in vitro* before processing. A split thickness skin biopsy of about 1-5 sq cm is harvested with all aseptic and antiseptic precautions from unburnt site of the patient as soon as the patient i to a burn center. The removed skin piece is tran tissue culture medium with antibiotics.

Keratinocyte Isolation

The harvested skin is thoroughly washed off th and subjected to differential trypsinization. The epidermal component is centrifuged and cell sus obtained.

Technique of Culture

The basic culture procedure described by Green e is followed at majority of centers. It is now a culture keratinocytes on feeder layers to obtain cultured epithelium sheets. This feeder layer usual of fibroblasts which are either locally irradiated or with mitomycin. Usually murine fibroblast line a human fibroblast are used as they allow similar g of keratinocytes.

The isolated keratinocytes are seeded on to pl dishes/culture flasks containing mitomycin C trea fibroblast 3T3 cells. The 3T3 cells are attenuated with mitomycin C and frozen in liquid nitrogen required they are thawed, counted and pla keratinocytes are grown in 3:1 mixture of D modified Eagle's medium supplemented with I medium, 0.4 mg/ml of hydrocortisone, insulin, 5 transferrin, 8×10^{-4} mmol/l adenine, 2×10^{-2} n iodothyronine, 10^{-10} mmol/l cholera toxin, antib 10% fetal calf serum. The cultures are incubated humidified atmosphere with 5% CO_2. After 3 days 10ng/ml of human epidermal growth factor (EGF to the medium. EGF acts when the differenti stratification begins in the center of the colon promotes the migration of keratinocytes to the Cholera toxin and cyclic – AMP activators also in cellular growth rate. Once a good primary established the keratinocytes are passaged 'n' times depending upon the required graft area.

The keratinocytes adhere to the extracellul synthesized by the irradiated/treated fibroblasts ar three types of clones: holoclones, merocl paraclones. The basal cells are considered as sten grow as holoclones and have the maximum growtl allowing 5-6 subcultures. Suprabasal cells paraclones and have the lowest growth potenti the cell culture, the holoclones progressively become meroclones and paraclones. As a result only second and third passages are used for grafting. Each colony of the cultured cells expands by the peripheral cells dividing and moving outwards. After 8-10 days when cultures are nearly confluent, all the cultures are seeded/reseeded with attenuated 3T3 cells.

The cultures are maintained by the same conditions until confluence and differentiation into multicellular epithelium is obtained (Figs 19.1 and 19.2). The epithelium is enzymatically detached from the culture flask or dish by Dispase and rinsed in PBS. The epidermis is taken on to Vaseline gauze of smaller mesh size by preserving the orientation of the graft in such a way that the basal cells are applied on to the wound bed (Figs 19.3 to 19.5). Cultured epithelial autografts usually take about 2-3 weeks time. The detached epithelium is assessed for cell viability (Green et al. 1979).[5]

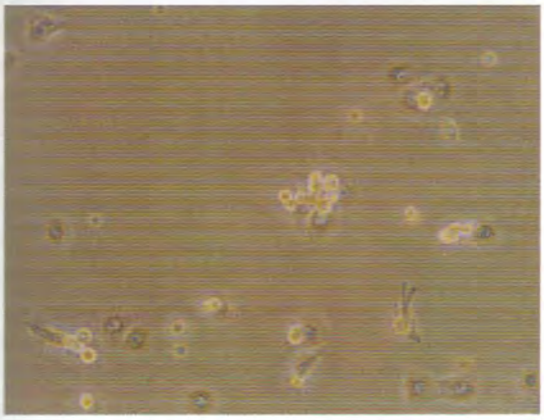

Fig. 19.1: Cell suspension of keratinocyte

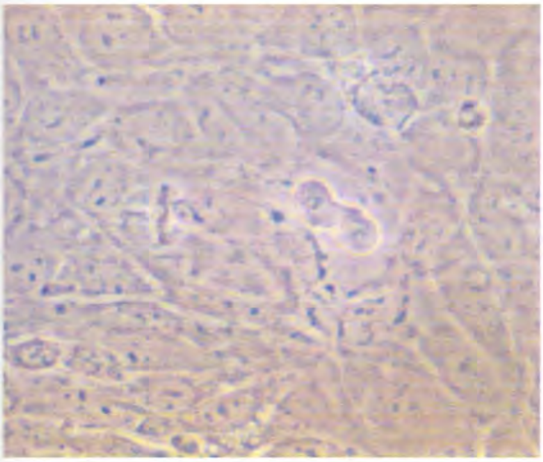

Fig. 19.2: Confluent keratinocyte on culture

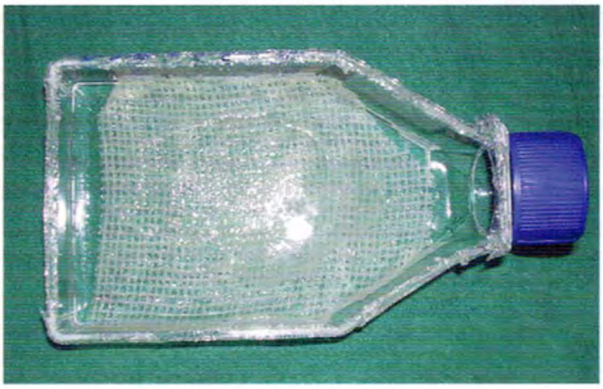

Fig. 19.3: Sheet of cultured keratinocytes on vaseline gauze before transfer

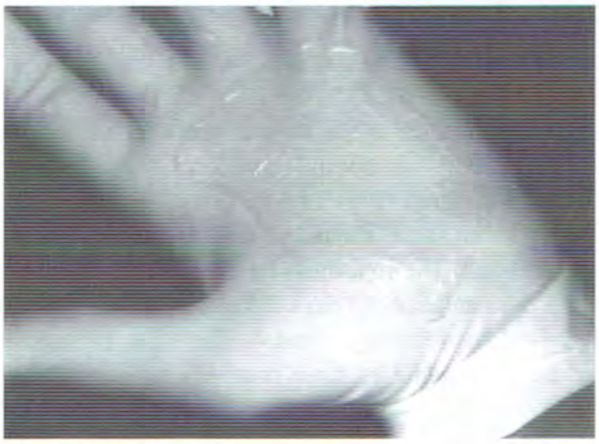

Fig. 19.4: Cultured keratinocyte sheet spread out on hand

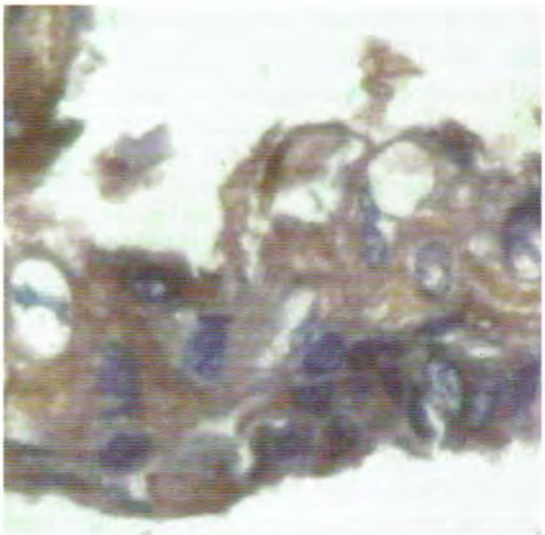

Fig. 19.5: Multilayered cultured keratinocyte under high power magnification

During this intervening period when the keratinocytes are being cultured, the patient's wounds are serially debrided to remove all the dead tissues after the patient's general condition stabilizes, usually after the third day post burn. The excision is done every 2-3 days and can vary from tangential to fascial depending on the depth of burns. The excised areas are covered either with a dermal skin substitute like biobrane or integra or if possible by cadaveric homograft.

The cultured graft material is transported in aseptic conditions in petridishes containing DMEM under 5% CO_2 to the operation theater, where the patient is prepared for surgery.

Grafting Procedure and Post Grafting Care

The grafting is carried out as soon as the cultured graft material arrives in the OT. The graft material with carrier gauze is applied on to the wound bed after debridement without friction, air bubbles and hematoma formation underneath the graft (Figs 19.6 to 19.8). The carrier gauze is covered with saline soaked gauze followed by thick dry gauze dressing. The first dressing change is done after the initial 5 days and every 2 days thereafter. The carrier gauze is removed between day 6 and 8. The other management procedures are same as those routinely followed for skin grafting. Limitation of cultured epithelial autografts for full thickness loss is the failure of this approach to reconstitute dermal layer. Epidermal replacement also ignores the fundamental importance of the dermis in providing the skin with its integrity and durability. Clinical problems of poor durability, scarring and wound contraction often are encountered. Graft 'take' can be improved by placing cultured keratinocytes autografts on a pre-grafted cadaver dermis.

Disadvantages

Many of the drawbacks associated with cultured keratinocyte autografts can be attributed to the absence of dermal element. The keratinocyte culture contains no dermal matrix tissue and is placed on debrided full thickness burns that lack any dermal elements. Since it is the dermis that provides the elastic quality to the skin, healed wounds that lack dermis are thin and stiff. Cultured keratinocyte grafts placed directly on the muscles or subcutaneous tissues form the basement membrane very gradually. The basement membrane serves as an important attachment function and epidermis lacking a well formed basement membrane,

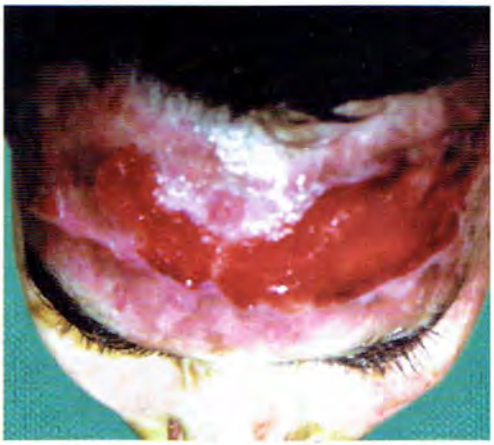

Fig. 19.6: Burn wound on forehead—Preoperativ

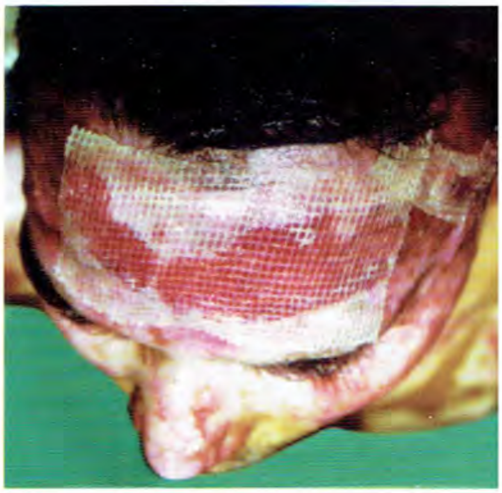

Fig. 19.7: Healed burn wound on forehead covered with keratinocyte sheet after 2 weeks of application

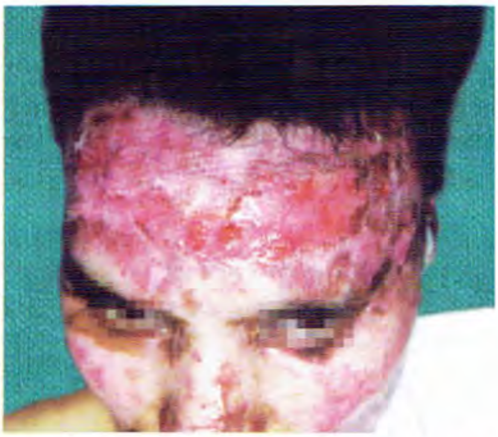

Fig. 19.8: Burn wound on forehead covered with cul keratinocyte sheet

blisters and shears off easily. Graft fragility results in 'delayed losses'. However, even a partial 'take' of epidermal cultured grafts can lead to complete wound healing by gradually progressing epithelialization from the islets. In the long term, it has been observed that these grafted areas tend to contract in the same manner as an autograft, though at the same time, the inflammatory reactions are weak and hypertrophic scarring rarely develops in these areas. The above drawbacks have limited their clinical applications and emphasize the need to develop keratinocyte culture methods on surfaces that mimic the human dermis or dermal equivalent.

There are reports of using various dermal equivalents with different compositions.[16-19] Even though they do increase the possibility of graft 'take', the high cost of these materials preclude their use for extensively burned patients who would require a large skin resurfacing in a short period of time. There have also been a lot of studies on using acellular fibrin gels as a biological support medium for keratinocyte cultures.[20,21] It has been observed that the keratinocytes cultured on fibrin gels are easier to handle and can be easily detached when prepared without using any enzymes so that the continuity of the layer of cells is preserved as use of enzymes reduces the adhesion potential of the cells. Also this method reduces the culture time to 15 days for producing sheet grafts. Currie et al[22] have also reported the use of keratinocyte cell sprays though the culture time remained at 5 weeks.

Epidermal Allograft Culture

Several centers have used cultured keratinocyte allograft to treat burn wounds with varying success. The cultured epidermal allograft can be available at all times and are much less expensive to produce. However, they undergo rejection similar to cadaveric homograft. The cells work best as temporary coverage where a supply of autologous keratinocytes is available to replace the allograft after rejection. The concept is that it will prevent the desiccation of underlying wounds and there is release of unknown growth stimulating substances by the cells as they die which will stimulate native wound healing.

SUMMARY

When Rheinwald and Green described the cultivation of strains of human epidermal keratinocytes with the formation of colonies of these cells from one single cell, there were claims by scientists that it had revolutionized the burn care of the future since it provided quick and easy cover of burn wounds following excision. Unfortunately this is far from reality as it is clear that cultured epidermis can in no way replace the classical autogenous skin grafting. Though it was thought that autologous keratinocyte culture was a boon for coverage of an extensively burnt patient but it is not universally accepted because of many of the imperfections associated with it, which are mainly unstable wound coverage, high cost, time consuming and sophisticated technology. It requires a close co-operation between the burn surgeons as well as cell culture biologists and all other personnel involved in patient care. The absence of dermal component produces frequent disruptions of overlying epithelium. Therefore, efforts to find suitable dermal component to be used along with epidermal cells are underway. Once the limitations of this technology are overcome by future research, acceptability of the same will increase manifold. At the same time the concept of tissue engineering using 'stem cells' is being explored and researched and if successful would have a dramatic impact on burn wound care and it may even take over the skin culture in the near future. However, further in depth studies will only tell about the real clinical potential of the method.

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Chapter

20

Burn Wound Infection

VK Tiwari, Sujata Sarabahi

INTRODUCTION

The management of patients with extensive burns has improved drastically over the years, greatly improving their survival rate. Yet microbial infections and resultant complications are the most important factors in burns mortality. Extensively burnt patients are immunocompromised and far more susceptible to infection than normal individuals despite the use of systemic and topical antimicrobials. Burn wounds at the onset are considered sterile. The amount of heat sufficient to cause damage to skin kills the microorganisms in and around the burnt area as well. Colonization by patient's normal skin flora, i.e. *Staphylococcus aureus* and *Streptococcus pyogenes* occurs within 48 hours following a burn injury as these organisms are located deep within the sweat glands and hair follicles surviving the thermal insult. Subsequent contamination of wound either by endogenous (gut flora) or by exogenous sources occurs shortly thereafter, usually within 5-7 days.[1-3] Blisters and eschar represent superficial and deep burn respectively and are part of dead and devitalized tissue and they are potential sites for colonization and bacterial invasion. The pattern of bacterial and fungal colonization is related in part to the topical or systemic antibiotic(s) used for prophylaxis and treatment of infection and varies with the area burnt. For example, burns of lower limbs are more frequently contaminated with enteric flora.

ETIOPATHOGENESIS OF BURN WOUND INFECTION

Simple colonization of the burned surface does not necessarily indicate infection but it can be dangerous as it can be followed by rapid proliferation of bacteria which eventually invade healthy tissues. Though the bacteria proliferate at the interface between the burn eschar and the underlying healthy normal tissues, invasion of latter manifests only at concentrations of 10^5 organisms/gm of tissue. With tissue invasion, local arteritis and thrombosis occurs producing characteristic satellite lesions around the burn wound. Septic emboli eventually also enter the blood stream producing abscesses in the liver, kidney and brain. The blood borne bacterial dissemination can be documented through positive blood cultures although this is true for gram-positive bacteria only. The absence of bacteria in blood cultures with clinical signs of sepsis indicates gram-negative toxemia. This is due to exotoxins liberated from the primary site of infection rather than the circulating bacteria per se.[4]

Older literature emphasized on Streptococcus and Staphylococcus as the most commonly isolated organisms in the burn wound. However, the frequency of these infections were gradually exceeded by gram-negative infections including those due to *Pseudomonas*, *E. coli*, *Klebsiella*, *Enterobacter*, *Citrobacter*, *Proteus* and *Bacteroides*.[5] With increasing use of topical and systemic antimicrobial agents over the past five decades, the fungal infection of burn wounds has becoming a growing problem and even viral infections have also gained significance. *Staphylococcus aureus*, especially MRSA (methicillin resistant) is again emerging as the most common cause of burn wound infection in many centers at present.[6-9] *Pseudomonas aeruginosa* has been reported to be the second most frequent agent followed by *Acinetobacter*, *Klebsiella*, etc. One of the most dangerous aspects of post burn infection by *Pseudomonas* and *Acinetobacter* spp. is the multi-drug resistance (MDR) of these organisms which is fast becoming a therapeutic problem. Even the bacteremia caused by gram-negative organisms is associated with a high mortality in burn patients.[10]

The incidence of invasive infection depends on the type of organisms present in the eschar.[11,12] *Pseudomonas* produces enzymes and other metabolic products and have flagella which enhance their potential for invasion and rapid spread. On the other hand, gram-positive organisms form different sized micro-abscesses in the eschar and subcutaneous tissues but rarely invade more deeply. *Candida* sp. also rarely invades deeper tissues. On the other hand, filamentous fungi like *Aspergillus* are more aggressive invaders of the subcutaneous tissues.

The most distinct pathologic response in burn wound is demonstrated in *Pseudomonas* infection which evolves from intra-eschar colonization to subeschar invasion. The diagnosis may be suspected when areas of hemorrhagic discoloration are seen in and around the burn wound though the actual infective focus may be hidden by the thick eschar. On histopathology of necrotic hemorrhagic tissues, it can be seen invaded by scores of bacilli. The reason for these lesions is attributed to vasculitis and effects of bacterial endotoxins. Typical colonization of bacilli seen in the walls of the blood vessels in case of *Pseudomonas* infection is far in excess of what is seen in infection with *Proteus* and other gram-negative bacteria.

MICROORGANISMS AND THEIR SOURCES IN BURN WOUND CONTAMINATION

Microorganisms can contaminate a burn wound from either endogenous or exogenous sources.

A. **Endogenous**—Bacterial source is patient's own body.
   1. Patient's normal skin flora –*Streptococcus* and *Pseudomonas*
   2. Upper respiratory tract—*Staphylococcus*
   3. Faecal flora—*E. coli* and other enteric bacteria.

B. **Exogenous** can be from:
   1. Community acquired infection—This is caused by *Streptococcus* and other less virulent bacteria that are sensitive to first line of antibiotics.
   2. Hospital acquired (Nosocomial) Infection— Existing bacterial flora of ICU/ Ward contaminates the burn wound. *Staphylococcus* (MRSA), *Pseudomonas*, *Acinetobacter* and *Klebsiella* are the common organisms which are usually resistant to the first line of antibiotics. The nosocomial transmission of microorganisms to the burn wound c
   transfer from the hands of health care pe
   through immersion hydrotherapy[13,14] o
   contaminated surfaces such as patient's r
   blankets, etc. In many of the burn units, i
   hydrotherapy treatment was a standard p
   the 1990's. It was found to increase t
   nosocomial infections and cross infectio
   the burn patients. Thereafter, showering w
   held sprayer gradually replaced hydrother
   for cleaning and debridement of burn wo
   has diminished the risk of transfer of bac
   one patient to another.

The various routes for invasion of pathogen
wound are as follows:

1. Burn wound: The burn wound with its exu
   necrotic tissue makes an excellent nutrient medi
   favors growth of microorganisms. Because of
   surface area involved in major burns, it is c
   susceptible to contamination by bacteria
   environment as well as patient's own endogen
   till the healing is complete. Patients suffer
   electrical burns with massive muscle necrosi
   produces an anaerobic environment, tend to ge
   with *Clostridium tetani* and *Clostridium perfrin*
   may manifest the disease frankly. With regul
   tetanus immunization in the pediatric age g
   following trauma, the incidence of tetanus
   reduced greatly.
2. Pulmonary: Infection of the lower respirator
   an important cause of death from sepsis in bu
   is common in patients with inhalational injury, e
   those requiring endotracheal intubation or trach
   and patients who do not receive prope
   physiotherapy.
3. Indwelling cannulae: Intravenous cannulati
   venesections, if not carried out with proper ase
   antiseptic precautions or if performed through t
   skin or if kept for a prolonged period of time, n
   to suppurative thrombophlebitis and multiple al
   along the length of the vein. This is an importa
   for sepsis as well as sudden thromboembolic d
   burn patients. The superficial cutaneous periphe
   of the burned areas represent a strategic route v
   focal invasion quickly leads to disseminated
   Therefore, veins cannulated beneath or adjacent
   wounds are more readily infected than tho
   unburned areas.

4. Urinary tract: Prolonged catheterization, which very often is unnecessary, can lead to urinary tract infection. However, prolonged catheterization may be imperative in children and female patients to prevent soilage of dressings on thighs and perineum.
5. Gastrointestinal tract: Translocation of bacteria through the gut is a very frequent occurrence in burn patients who have not been resuscitated well and are hypovolemic for a long time after sustaining burns. This is due to the sustained splanchnic vasoconstriction leading to gut mucosal erosions and migration of bacteria and their toxins through the gut wall into the peritoneum. This is an important cause for sepsis in burn patients.
6. Miscellaneous: Escharotomy incisions, burned areas which involve cartilage, e.g. the burns of pinna, are particularly susceptible to infection. Suppurative chondritis, endocarditis, arthritis also form a focus of infection in a burn patient which can, in rare cases, lead to sepsis.

TYPES OF ORGANISMS IN BURN WOUND CONTAMINATION

Since 1960 till date, *Pseudomonas aeruginosa* has remained the commonest organism isolated from the burn wound. Prevalence of this organism is around 60% in most of the burn units. Average distribution of different microorganisms is given in Table 20.1.

TERMINOLOGY USED IN BURN WOUND INFECTION

There are many terms used while defining different stages in burn wound infection. They are as follows:

Colonization

Surface culture from burn wound shows presence of pathogenic organism and count is $<10^5$ organisms / gm of surface tissue. This is also termed as supra-eschar colonization. This supra-eschar colonization through dead hair follicles, may invade eschar tissue usually termed as non-invasive wound infection.

Non-invasive Wound Infection

Infection is limited to eschar and sub-eschar tissues. Bacterial count in eschar may be $>10^5$/gm of tissue. Sub-eschar infection helps in early eschar separation. In extensive burns and low resistance individuals this sub-eschar colonization may lead to invasion of healthy tissues and burn wound infection.

Burn Wound Infection

Invasion of normal non-burned tissues by microorganisms is known as burn wound infection. Quantitative bacterial count is $> 10^5$/gm of tissue.

Bacteremia

Presence of viable bacteria in blood is known as bacteremia.

Septicemia

Systemic illness caused by the spread of microbes or their toxins via the bloodstream is known as septicemia.

Systemic Inflammatory Response Syndrome (SIRS)

If two of the following four signs are present, the patient is labeled as having SIRS.

1. Oral temperature is >38 °C or is < 36 °C.
2. Respiratory rate of >20 breaths/minute or $PaCO_2 < 32$ Torr.
3. Heart rate > 90 beats/minute.
4. Total leucocytes count $>12000/mm^3$ or $< 400/mm^3$

Sepsis

SIRS with proven microbial etiology in any patient is termed as sepsis.

Sepsis Syndrome

Sepsis and one more sign of organ dysfunction like hypertension, metabolic acidosis, alteration of mental status, oliguria, ARDS or Platelets count < 0.1 million/mm^3.

Multiple Organ Dysfunction Syndrome (MODS)

Dysfunction of more than one organ requiring intervention to maintain homeostasis like ventilatory support for ARDS and dialysis for renal failure, etc.

The microbial status of burn wound can be further classified according to the depth of involvement into:

Stage I is the *stage of Colonization*—

a. Superficial colonization: Organisms are present only on the superficial surface

Table 20.1: Showing the various types of microorganisms isolated from burn wound, their incidence and sensitivity in the authors' unit

Type of organisms	*Name*	*% Incidence*	*Source*	*Antibiotic susceptibility*	*Remarks*
Gm +ve Cocci	*Staphylococcus* MSSA	1 to 2	Community acquired	Amoxicillin and Cephalexine	Present in burn wound of patients treated at other hospital for 5 to 7 days before coming to burns un
	Staphylococcus MRSA	10 to12	Nosocomial	Vancomycin and Teicoplanin	Cause of fulminant sepsis in extensive burns
	Streptococcus β Haemolyticus	0.5 to1	Endogenous	Crystalline Penicillin	100% graft failure if present in bu wound
	Streptococcus Faecalis	0.5 to1	Endogenous	Crystalline Penicillin	In immunocompromised extensiv burns patient, can cause fulminan sepsis
Gm. –ve Bacilli	*Pseudomonas Aerugenosa*	65 to 70	Nosocomial	Beta Lactamase producing Sensitive to Cefperazone + Sulbactam and Piperacillin + Tazobactam. ESBL Sensitive to Carbapenems (second generation)	Rapidly develops multi-drug resist by efflux pump mechanism
	Klebsciela	10 to 12	Nosocomial	Gr.2 Beta Lactamase and Extended Spectrum Beta Lactamase producing, Sensitive to Cefperazone + Sulbactam, Piperacillin + Tazobactam, Amino glycosides and Carbapenems (second generation)	More likely to cause fulminant lung infection
	Proteus	2 to 5	Nosocomial	Gr. 2 Beta lactamase producing, sensitive to Amino glycosides, Cefperazone + Sulbactam, Piperacillin + Tazobatum, Carbapenems	Usually respond to proper timely intervention
	Acinetobacter	2 to 5	Nosocomial	ELBS producing, Sensitive to Carbapenems (Also known as Gm –ve bacilli equal to MRSA)	Fulminant pulmonary complicati
Fungal infection	Candida sp.	50 to 60	Endogenous	Azoles, Caspofungin	Mainly in patients on prolonged antibiotic combinations, TPN, dia

b. Microbial penetration: Microorganisms invade the eschar
c. Subeschar proliferation: Microorganisms proliferate in subeschar space

STAGE II is the *stage of invasion of viable tissue*—

a. Microinvasion: Microscopic foci of organism in viable tissue below burn wound
b. Generalized invasion: Diffuse penetration into viable subcutaneous tissue
c. Microvascular invasion: Organisms invade into l blood vessels and lymphatics

IMMUNE PROFILE OF EXTENSIVELY BURN PATIENT[15-20]

The natural resistance of human body to infection kind of wound is primarily a function of the innate in system. Following burns, the innate immune s responds by stimulating localized and systemic inflammatory reaction. But in extensive burns, patients are usually immunocompromised due to:

a. Alteration in the function of neutrophils, lymphocytes and macrophages.
b. Alteration in Immunoglobulin kinetics.

This results in depression of both cellular and humoral immunity. As a result, the burn victim gets predisposed to infectious complications. The host response to thermal injury is mediated to some extent by arachidonic acid and cytokinin cascade and the combined effect of this cascade being release of various endotoxins, like Prostaglandin E_2 (PGE), Thromboxenase, α_2-vasoconstrictor, TNF (Tumor necrosing factor) etc. The alteration in the immune system has a combined effect of increasing the burn patient's exposure to pathogens and decreasing the natural defenses responsible for counteracting them. Because of impaired function of the neutrophils, natural killer cells and macrophages, the body's immune system is unable to eradicate the pathogens to which the wounds are exposed. Thus, burn patients are more susceptible to wound infections, sepsis and multi-organ failure. Even the reduction of levels of circulating immunoglobulins like IgG and IgM have been associated with high burn wound infection rate and mortality. Briefly, there is a marked diminution of serum IgG concentration, total and all subclasses. The maximum fall occurs at 2-3 days after injury when the mean concentration of IgG, IgA, IgM, IgD and IgE have been shown to be 30, 44, 49, 35 and 22% respectively of the level found in healthy controls. Their concentration returns to normal level after 19, 9, 7, 30 and 11 days respectively.

CLINICAL FEATURES

Early Diagnosis of Burn Wound Infection (Figs 20.1A to 20.3)

Local Signs of Burn Wound Infection

- Black or dark brown focal areas of discoloration.
- Increasing eschars
- Increase in the depth of burn injury.
- Increasing inflammation and edema in normal skin adjacent to burn margin.
- Increased bleeding from granulation tissue during dressing.
- Rashes or papule in skin surrounding burn wound and/ or distant part of body.

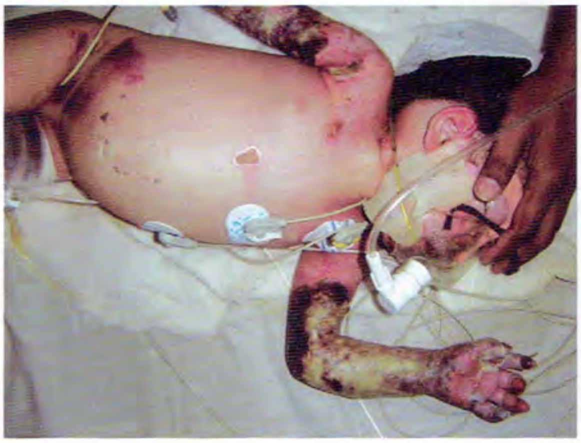

Fig. 20.1A: A 2-year-old child with 20% burns with increasing eschar indicating increasing depth of burns with increasing inflammation and edema in normal skin

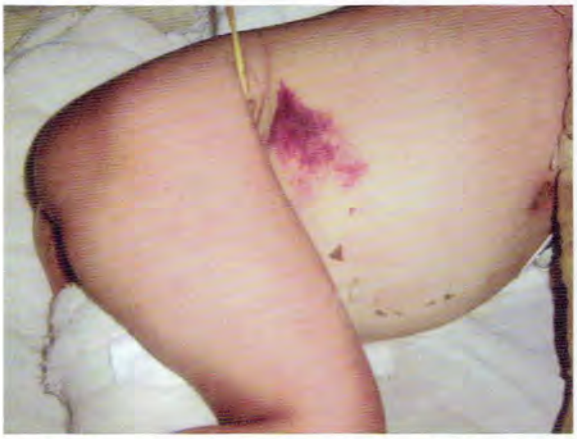

Fig. 20.1B: Same patient with hemorrhagic patches with edema of uninvolved skin indicating patient is landing up in DIC (Disseminated intravascular coagulation)

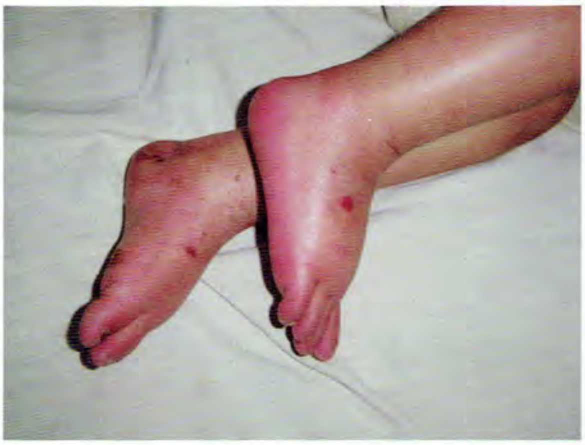

Fig. 20.1C: Same patient with rashes, papules and erythema in non-burned extremities indicating invasive gram-negative sepsis

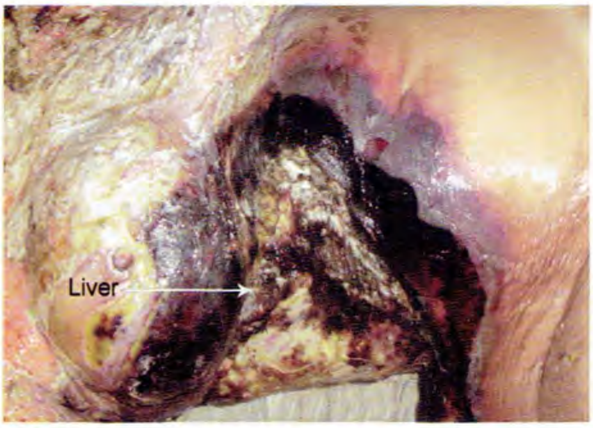

Fig. 20.2A: Synergistic gangrene of abdominal wall following uncontrolled burn wound infection. The chest wall and abdominal muscles are totally necrosed which is likely to expose the underlying structures

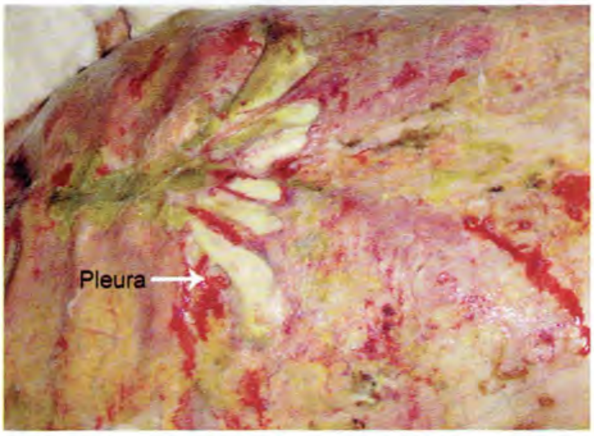

Fig. 20.2B: Neglected patient showing deepening of burn wound up to ribcage with exposed pleura

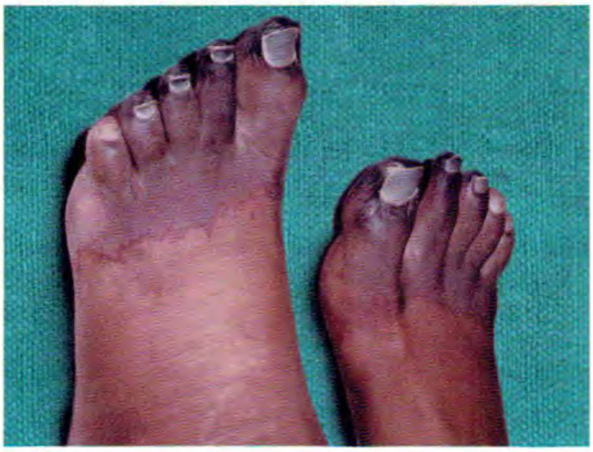

Fig. 20.3: Patient in gram-positive sepsis throwing septic emboli causing bilateral gangrenous changes in distal foot

Systemic Signs of Burn Wound Infection

1. Signs of gram-negative septicemia –Rapid o symptoms within 8-12 hours characteriz hyperthermia (100-103°F), tachycardia and tach Patient is well perfused with bounding pulse. S blood pressure is initially normal or raised (high shock). Pulse pressure is usually varied as vasodil may reduce diastolic pressure to a considerably lo Later on, if sepsis remains untreated systolic pr also drops (low output shock). Mental cloudin diarrhea are sometimes presenting symptoms o wound infection leading to sepsis. Multiorgan fail late stage may involve any system causing renal f paralytic ileus, Acute Respiratory Distress Syn (ARDS). Jaundice and disseminated intrava coagulation (DIC) are very late manifestation usually have very poor prognosis. Decrease in pl counts is another diagnostic sign of beginning of s
2. Signs of gram-positive septicemia—Symptom gradual and usually start with hyperpyrexia (t >105°F) and leucocytosis. Hypotension, paralytic and oliguria are common with initial gram-po septicemia.

The most reliable clinical sign of an invasive burn w infection is the conversion of an area of partial thic injury to full thickness necrosis (Fig. 20.4). Any chan wound appearance mandates immediate biopsy of the for histopathology and culture sensitivity as wound bi is the only reliable way of differentiating micr colonization from invasive infection. Beta hemo streptococcal infection may be manifested by an inc in erythema at the margins of the burn wound, like in cellu with inflamed and edematous burn wound (Figs 20.5 20.6).

Colonization of burn wound by *Pseudomona* evidenced by bluish green discoloration of the wound w is due to pyocyanin or there can be erythematous, violac or necrotic escharified nodular lesions in the unburned called as Ecthyma gangrenosum (Figs 20.7A to C). W there is a foul smelling odor with purulent discharge, Pro organism should be suspected.

Following excision of a burn wound, if the surfa left exposed, the wound surface undergoes desicca which creates a neoeschar. This should not be mistake infection-related necrosis. Therefore, excised areas sh always be covered immediately either by autogra homograft.

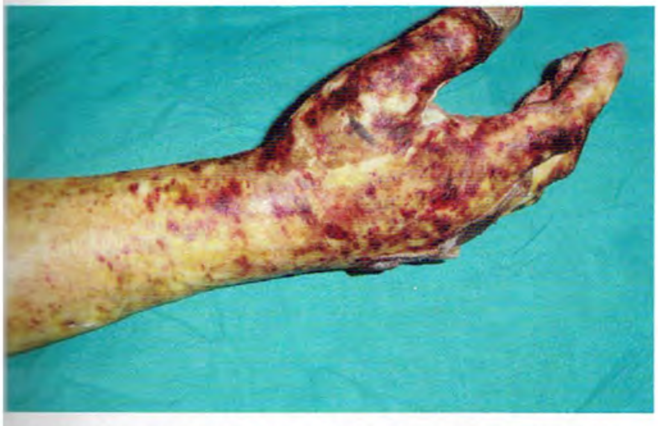

Fig. 20.4: Superficial burns converting to deep burns indicating burn wound infection—Black areas of discoloration, thickening eschar

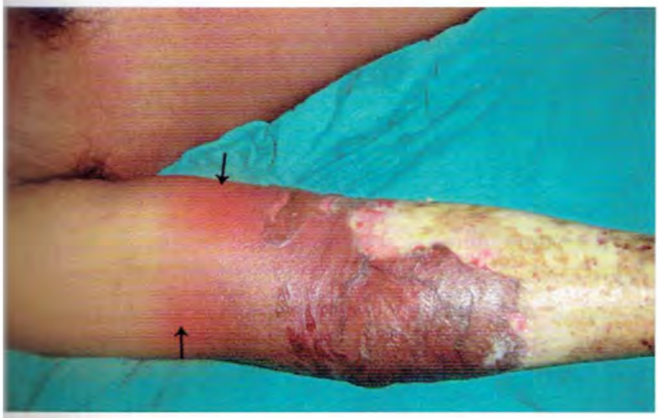

Fig. 20.5: Streptococcal burn wound infection with extending erythema into surrounding normal skin from burn wound margin

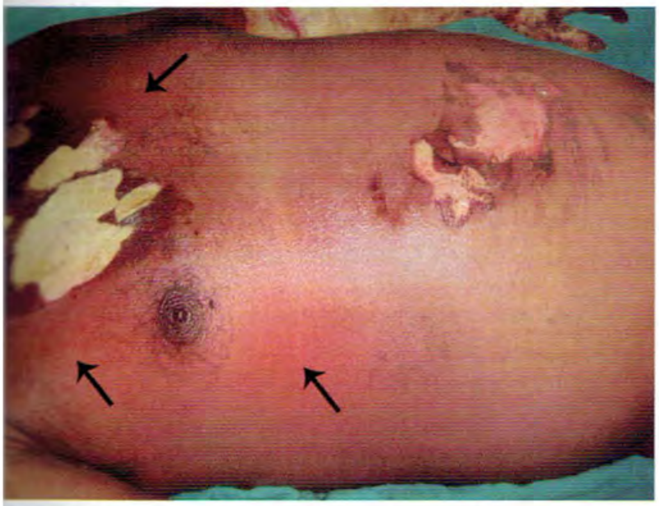

Fig. 20.6: Another patient with streptococcal erythema involving unburnt skin of chest and abdomen

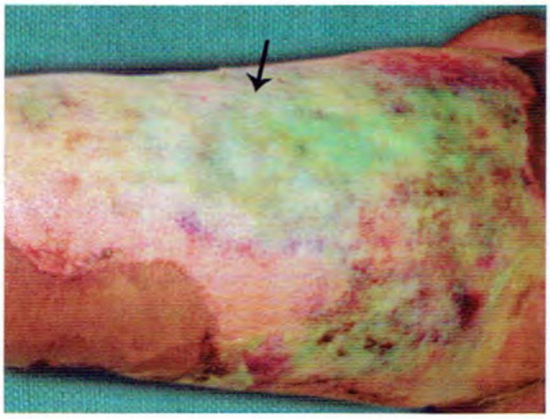

Fig. 20.7A: Greenish discoloration of the dressing and burn wound surface indicating *Pseudomonas* colonization of the wound

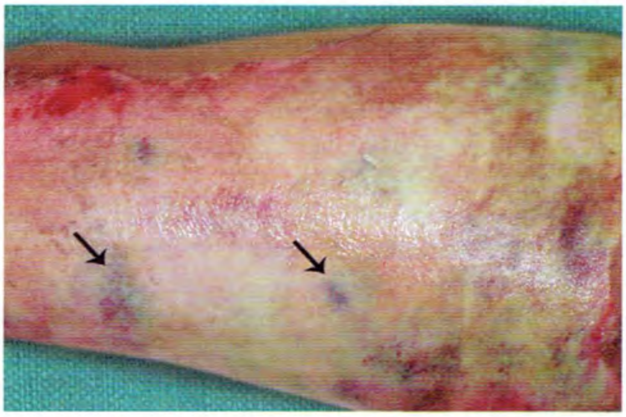

Fig. 20.7B: *Pseudomonas* invasion of the same wound indicated by bluish purple patches appearing in the depth of the burned area

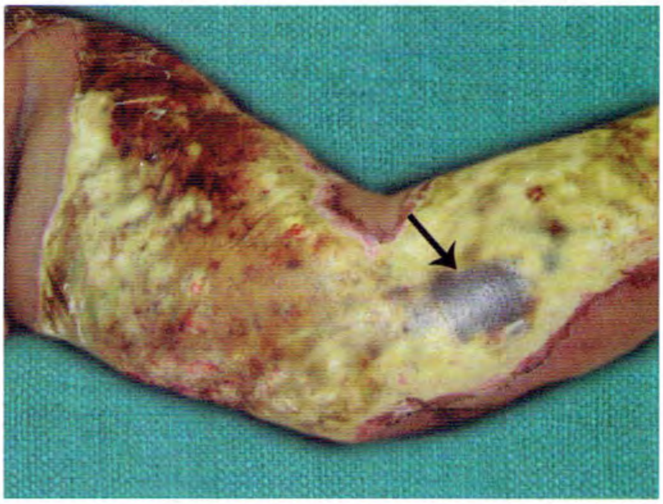

Fig. 20.7C: Full blown *Pseudomonas* invasive sepsis–Hemorrhagic patches with thickening eschars, increasing soakage from wound

FUNGAL INFECTION IN BURNS

Although fungal growths were noticed in burn wounds prior to the use of topical antimicrobial therapy, with their extensive use for burn dressings, the incidence of fungal colonization in burn wounds has increased. In fact after the introduction of Mafenide for burn dressing there was a four fold increase in the incidence of fungal burn wound infection.[21] This trend continued with incidence of bacterial infections decreasing over the years till such a period when fungi represented the most common burn wound infection.[22] The fungi most frequently isolated include *Candida*, *Mucor*, *Rhizopus*, *Aspergillus* and *Alternaria* sp.

The fungal involvement of burn wound is classified into fungal wound colonization (FWC) and fungal wound infection (FWI).

Fungal wound colonization (FWC) is defined as presence of fungus in the burn eschar without penetration to the level of viable tissues. Fungal wound infection (FWI) is defined as invasion into viable tissue below the eschar.

The fungi can be grouped into 3 categories:

a. *Aspergillus* like morphology
b. Mucor like morphology (Zygomycosis/ mucormycosis)
c. Yeast like morphology (*Candida*)

Candida albicans has been reported as the most frequent pathogenic species causing nosocomial fungal infection in burn patients with an incidence of 3%[6], with mortality ranging from 38 to 50%.[23,24] The incidence of candidiasis increases as the duration of stay in the hospital increases. It has been reported from the authors' unit that the incidence of candidiasis is 5% in less than 7 days post burn patients and it increases to 58% in more than 7 days post burn.[25] This is attributed to long term use of systemic antibiotics, central lines, use of total parenteral nutrition, immunosuppression and ventilatory support.

The earliest gross findings of fungal infection are hemorrhagic area(s) of discoloration in burn wound. Some of the fungi especially Mucor and *Aspergillus* have a preference for vascular invasion and therefore, produce widespread ischemic necrosis of soft tissues which may be so severe as to produce gangrene of an entire limb. Necrosis and saponification of subeschar fat beneath the burn wound is also characteristic of fungal infection and this is seen as a cheesy yellow appearance of subeschar tissues when the burn wound is debrided (Figs 20.8 and 20.9A to C). Histopathologic examination of wound biopsy is required for differentiating fungal infection from the other bacterial infection.

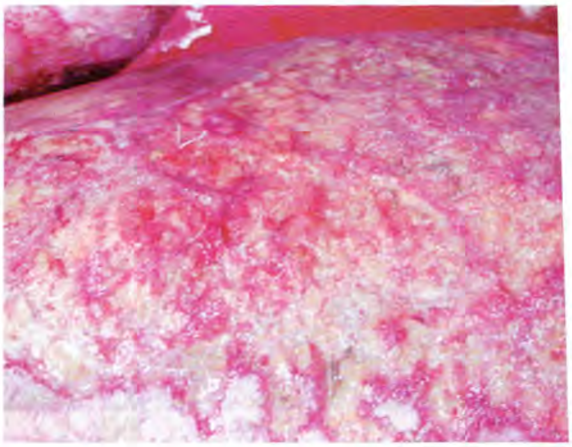

Fig. 20.8: Close up view of Invasive fungal sepsis du
Aspergillus infection

VIRAL INFECTION IN BURNS

Viral infection in burn patients has not receive
attention in the past but burn wound infection by
Varicella viruses and Cytomegalovirus has been rep
The reason given for this is the lowered immune s
the patient which allows the latent viruses to m
themselves. The lesions usually occur in the area of
second degree burns and present as vesicles and sup
erosions with secondary bacterial infection. Ext
cutaneous herpetic infection or varicella zoster (Chick
infection may develop in some burn patients and the sy
dissemination to liver, adrenals, respiratory and gastroin
system along with secondary bacterial infection of cut
lesions may be quite fatal (Figs 20.10 A to C). Only
of an early lesion will manifest signs of infection by H
Varicella group because rapid colonization by *Staphylo*
and *Pseudomonas* later can obscure the viral nature
lesions. Usually shave biopsy of entire vesicle pr
adequate tissue for diagnosis. Diagnosis can al
established by measuring neutralizing or complement
antibodies in acute and convalescent phase sera b
viruses cannot be isolated from older lesions.

MANAGEMENT OF BURN WOUND INFECTI

There are three levels of management of burn
infection:

a. Prevention of burn wound contamination from exo
source (Primary line of management).
b. Prevention of burn wound infection and septice
wounds which are already contaminate

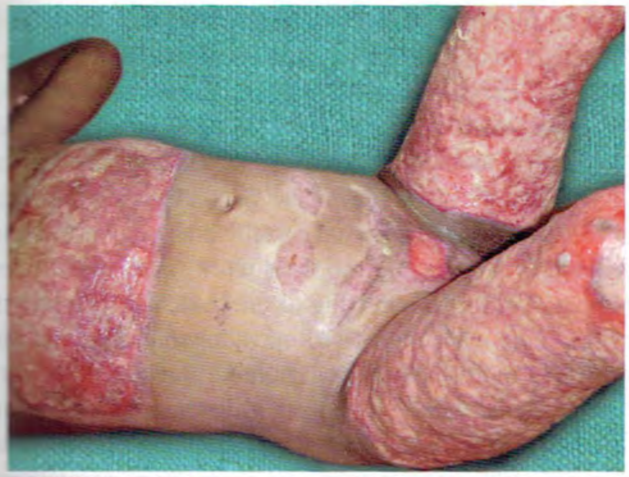

Fig. 20.9A: A 3-year-old child with fungal sepsis—Wounds are pale, edematous, nongranulating with exposed subeschar fat

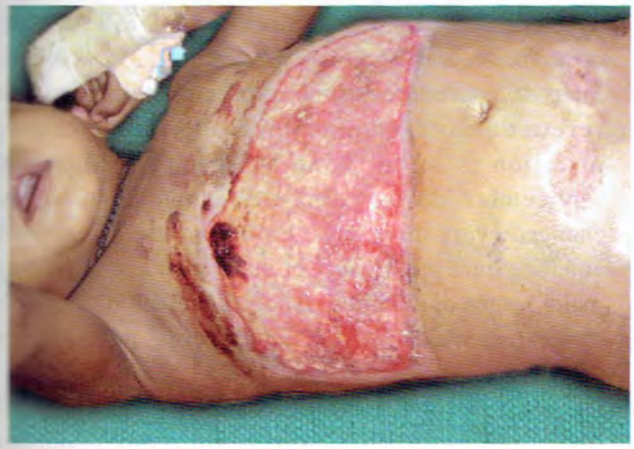

Fig. 20.9B: Same patient's typical septic look

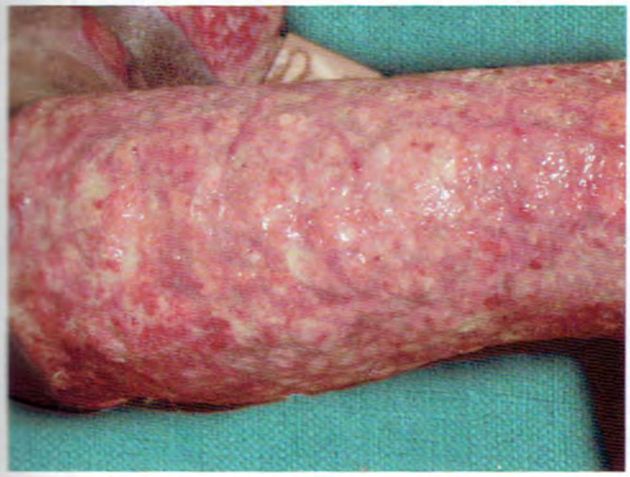

Fig. 20.9C: Close up of the same invasive fungal wound

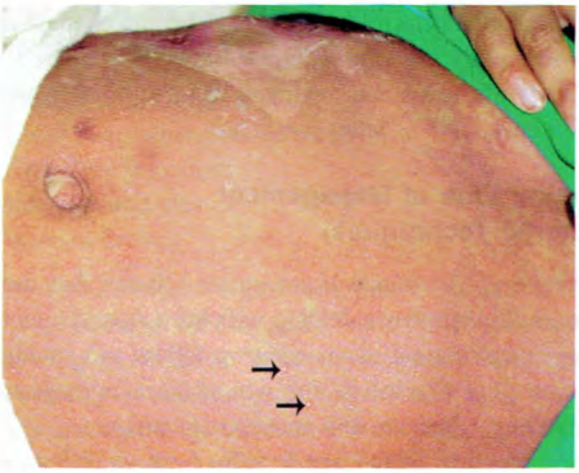

Fig. 20.10A: Typical varicella infective lesions in a healing burned patient

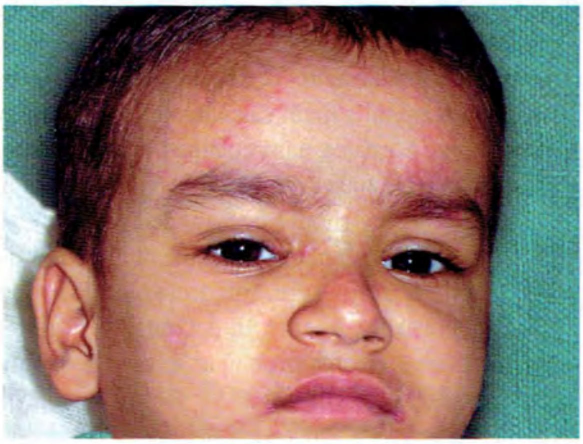

Fig. 20.10B: Same patient with vesicular lesions on the face—Varicella zoster infection

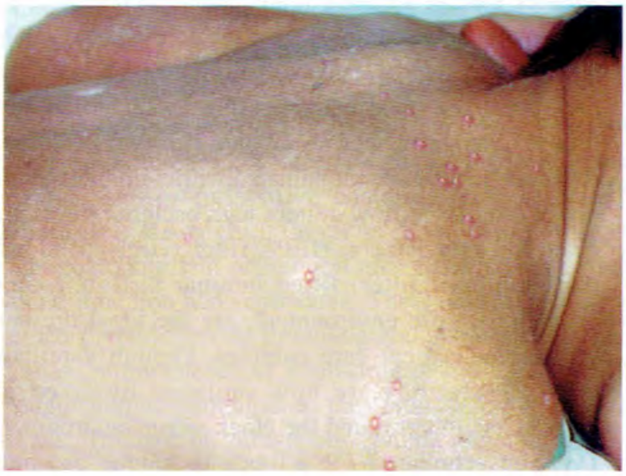

Fig. 20.10C: Distribution of lesions on the back in uninvolved skin

microorganisms from exogenous or endogenous sources (*Secondary line of management*).

c. Early diagnosis and treatment of infected burn wound (*Tertiary line of management*).

Primary Line of Management (Aseptic Techniques)

The saying, "An Ounce of prevention is better than tons of cure", holds true when dealing with burn wound infection. The principles are same as what we follow and practice in any surgical discipline for wound care management, comprising of aseptic and antiseptic techniques.

1. Primary line of management for burn wound starts as soon as the patient enters the casualty or even earlier at the site of accident. Wounds are essentially sterile at the time of burns. Prevention of wound contamination is, therefore, of utmost importance. The public should be taught to cover the burn wounds with cleanest sheet available. In the casualty, the first dressing should not be done in hospital's main casualty, unless the hospital has a separate burns casualty manned by proper trained staff. If extensive burns are being received in hospitals main casualty, they should be wrapped in sterile sheet with sterile McIntosh placed underneath the victim and transported to burns unit for dressing. Practice of applying coloring agents like Gentian violet and Mercurochrome creates problems in burn assessment; hence, should be discouraged.
2. Dressing stations—The dressing stations are an important site for transmission of infection from health care workers to the wound as well as from one patient to another. During dressing of any extensive burn, there is heavy contamination of the air inside the room. There should be a gap of at least 30 minutes between two dressings. The ideal burn dressing room should have total exchange of sterile air between two dressings. Room air can be decontaminated within minutes by the use of laminar airflow system with bacterial filters and fixed air aseptisizers. HEPA filter (High efficiency particulate air filter) fitted laminar airflow rooms, providing sterile environment, are the ideal dressing station and patient care cubicles. Plenum ventilated dressing stations are now replaced by fixed air aseptisizers to get round the clock sterile environment.
3. Dressing technique — "No Touch Technique" is a must in treating the burn wound. Burn dressing is a highly skilled job and should be carried out by the perso trained in that. Three teams are responsible for c of one dressing only, which includes doctors, n and dressers. First team which involves a nurs dresser is for dressing removal only and is usually k as "dirty" or "unsterile" nurse. Second team is the dressing team who does the sterile dressing work. team is responsible for floor work, known as " nurse". Once the dressing of one patient is compl the entire team changes for next dressing. Minimu two to three such teams should be there in a "he burns unit, where 8 to 10 extensive dressings are performed daily. In minimal touch technique instru are mainly used for doing dressings, burns wound not at all touched by hands. Priority of dressing sh be for clean, less extensive and pediatric dressings extensive and 'dirty' dressings to be done later. workers in the dressing room should follow s operation theater norms.
4. Invasive procedure like urethral catheterization venesections or CVP(central venous pressure) can insertion is often found to be major sourc septicemia. Ideally peripheral cannulae shoul changed every three days. Prolonged use of cath and long lines require proper maintenance for avoi major source of infection, especially infective deep thrombosis which often is the triggering point of septicemia.
5. Prevention of infection in Burn ICU—Extensive b patient require isolation as well as maximum nur care and monitoring in a burn ICU. Barrier nurs patient isolation and environmental sterilization are methods to prevent cross infection. All health personnel should be appropriately gowned and glo at each entry into the patient's room. Simple hand w during ward rounds, before and after seeing the b patient is the simplest way to prevent cr contamination. Bacillocid spray 30 minutes bef receiving the patient is equally effective and less t than fumigation. All equipment in each patient's ro should be regularly cleaned with appropri disinfectants.
6. Culture of environment of burn ward, burn ICU and other burn stations by exposing culture disc for minutes and culture from beds, monitors and ot equipments is done every 3 weeks . If found posi their remedial process is very important aspect of b

wound prevention. Formalin fumigation and carbolization of bed and equipment have largely been replaced by modern environment friendly safe procedures like Bacillocid spray of walls and equipments. Ultraviolet light can be installed to reduce bacterial count in vacant areas (without patient). Usually a combination of the following procedures is used to minimize environment and fomite contamination.

Procedures that predispose the patient to cross–contamination, such as hydrotherapy tanks, are kept to a minimum. Cohort nursing care is another component of environmental control in a burn unit. Nurses and other health workers are assigned to work as a team for one specific patient or a group of patients and the movement between patients is strictly limited. Patients convalescing are kept separate from the acute patients since they form a reservoir of more antibiotic resistant organisms which are acquired during their hospital stay. Thus, after the ICU, there is a step down ICU where the patient is shifted after he/she is fully resuscitated and stabilized and thereafter, there should be a chronic ward where patients are prepared for skin grafting of the raw areas.

a. Physical
   1. Laminar-air flow system with HEPA filters (high efficiency particulate air filters).
   2. Fixed or mobile air aseptisizer (Fig. 20.11)
   3. UV light exposures

b. Chemical
   1. Formalin fumigation, carbolisation, bacillocid spray.

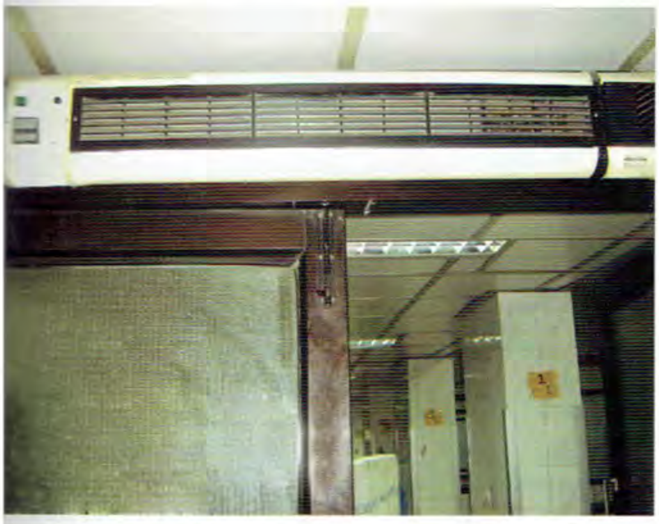

Fig. 20.11: Fixed air aseptisizer at the entrance of Burns ICU and wards to reduce bacterial count

Secondary Line of Burn Wound Management (Antiseptic Methods)

A. Prophylactic antibiotics
B. Local chemotherapeutic agents
C. Biological dressings
D. Early wound closure
E. Immunotherapy

Prophylactic antibiotics: Role of prophylactic antibiotics in burns is debatable. Healthy adults up to 30% burns may not require any prophylactic antibiotic. Extensive burn patients are usually immunocompromised and may require prophylactic antibiotics. A diabetic burn patient and the one coming late to a burn center from far-flung areas with open wounds have fair chances of wound contamination and invasion. Prophylactic first line of antibiotics has a definite role in such types of community acquired infection. Crystalline penicillin in combination with an aminoglycoside usually takes care of community acquired infections and *Clostridium tetani* as well, if patient is not previously immunized for tetanus.

Local chemotherapeutic agents: 1% Silver sulfadiazine cream for extensive burns usually takes care of surface contaminants or endogenous contamination from hair follicles with painless acceptable eschar penetration and minimal toxicity. Therefore, it is the ideal topical antimicrobial cream for extensive burns.

Biological dressings: Collagen dressings and other biological dressings seal the wound from external environment and thereby reduce the chances of wound contamination.

Excision of deep dermal and full thickness burns with immediate skin grafting reduces the morbidity. Elimination of the necrotic tissues and raw areas, in turn, reduces the chances of infection and septicemia.

Immunoprophylaxis: Apart from active and passive tetanus prophylaxis many other vaccines are available for gram-negative and gram-positive bacteria. Their efficacy is debatable. However, there is a definite role of immunoglobulins in prevention and management of burn wound infection and septicemia. The availability of much refined preparations of immunoglobulins and widespread increase in prevalence of multi-drug resistant bacteria has brought immunoglobulins to the forefront during the last decade. The role of intravenous gamma globulin factor in burn patients has been studied extensively in the past.[17,18] Recently, the use of IgM enriched immunoglobulins to enhance the humoral response has emerged as a valuable tool in the management of burn patients with infection. Experimental *in vitro* and animal studies on IgM enriched immunoglobulins have demonstrated that elevated titre of anti-endotoxin antibodies correlate significantly with decreased mortality due to gram-negative sepsis. Besides neutralizing antigen, IgM enriched immunoglobulin also activate compliment factor like opsonins. To enhance immune response against bacteria, IgM immunoglobulin has been shown to have a synergistic action with beta-lactam antibiotics as well as a beneficial immunomodulating effect.[19,20]

Tertiary Line of Management of Burn Wound Infection

Early Diagnosis and Treatment (Figs 20.12A to N)

Early diagnosis of burn wound infection and septicemia and specific treatment for the same is the key for decreasing mortality in extensive burns. Going by the definition of SIRS(systemic inflammatory response syndrome), almost all extensive burn patients are in SIRS. They have to be regularly observed for signs of septic syndrome. Patients with diabetes or other immunocompromised conditions are more likely to land in septic complications. Following local signs and symptoms, if present, are the starting point for invasive infection:

1. Unexplained pain or discomfort in burn wound
2. Excessive soakage
3. Unexplained fever
4. Inflamed burn wound margins are the starting point of wound infection. In facial burns especially if ears are involved, increased cephaloconchal angle is the first sign of inflammation.
5. Gastrointestinal symptoms in the form of loose motions and or abdominal distension
6. Abnormal behavior, intermittent irrelevant talks and oliguria despite adequate perfusion are sometimes the presenting features of sepsis.

Investigations

Investigations along with these preliminary signs and symptoms help in diagnosing the specific organism invading the burn wound.

1. Leukocyte count and platelet count –Importance of total leukocyte count in relation to infection is well documented. Leucocytosis in early sepsis and leucopenia in late sepsis is often diagnostic. Persistently decrea platelet count is often the first sign of impending se syndrome in extensive burns.
2. Invasive burn wound infection usually starts after 5th post burn day in extensive burns. Initial surf culture report, if available, is the best guide for the t of organism and its antibiotic sensitivity.
3. Blood culture is usually non-informative as 75% ca are in gram-negative septicemia in which only the tox are present in blood. In gram-positive septicemia bl culture report is usually more informative.
4. Wound biopsies –Quantitative wound analysis is help in some cases not responding to treatment as bacte counts more than 10^5 organisms/gm of tissue dema for a multipronged approach for burn sepsis. Ser biopsies are essential to follow the progress of infecti Any change from stage Ia to stage Ic (as mention earlier) demands immediate change in wound ca management. If stage II is diagnosed, excision of t eschar and underlying infected tissues should be carri out as soon as possible. If gram-negative invasion present, the topical antimicrobial agent being used stopped and mafenide cream should be used till excisi is performed. Wound biopsy is also indicated for fung infection, usually superimposed, once bacterial infecti is being successfully treated with advanced antibiotic

Treatment

Depending on extent of body surface area burnt, the typ of infecting organism and the general condition of the hos the treatment should be planned against the infectin microorganism using

a. Local and systemic antimicrobial agents
b. Increasing the host resistance to infection by usin Immunoglobulin
c. Enteral and parenteral nutrition including albumin an blood transfusions
d. Controlling concomitant diseases like diabete hypertension, etc.

Local Wound Dressings

Early burn wound infection requires change of antimicrobi agent to the one which is effective against the suspecte organism(s). Unfortunately, very few agents are availabl for use in extensive burns with good eschar penetration. 1% silver sulfadiazine (SSD) cream is being used an subsequently *Pseudomonas* infection is suspected, chang

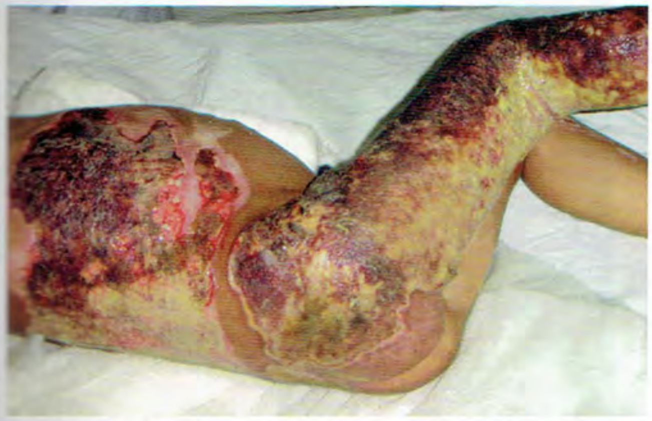

Fig. 20.12A: Patient with invasive gram-negative burn wound sepsis—Hemorrhagic discoloration of entire eschar

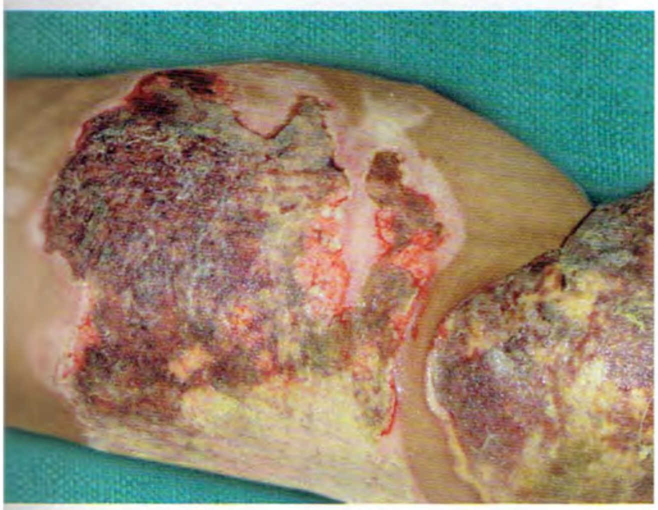

Fig. 20.12B: Close up view of same wounds

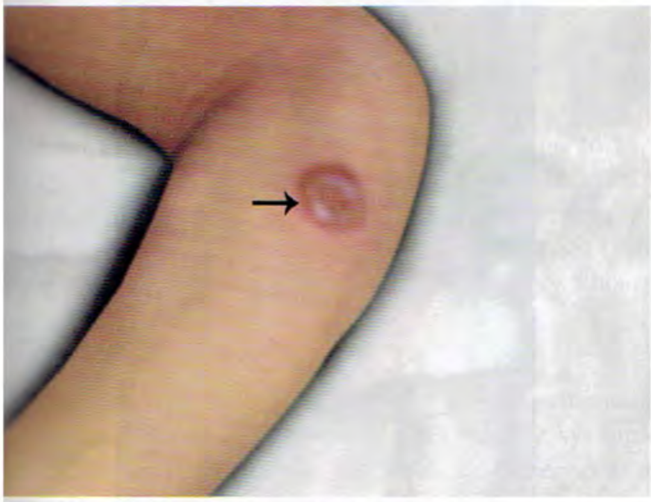

Fig. 20.12C: Typical nodular satellite lesions in uninvolved skin—Ecthyma gangrenosum

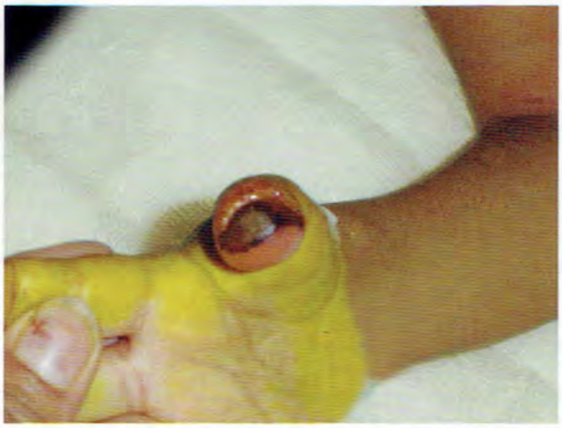

Fig. 20.12D: Infective gangrenous changes in thumb indicating septic embolization

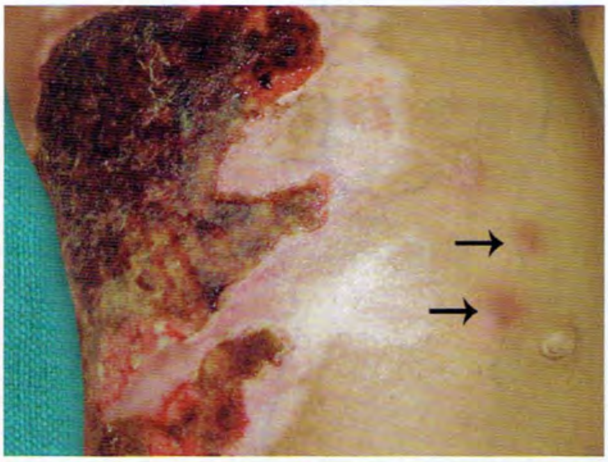

Fig. 20.12E: Nodulopapular lesions in adjacent unburned skin over the abdomen and chest with edema of the skin with distension abdomen

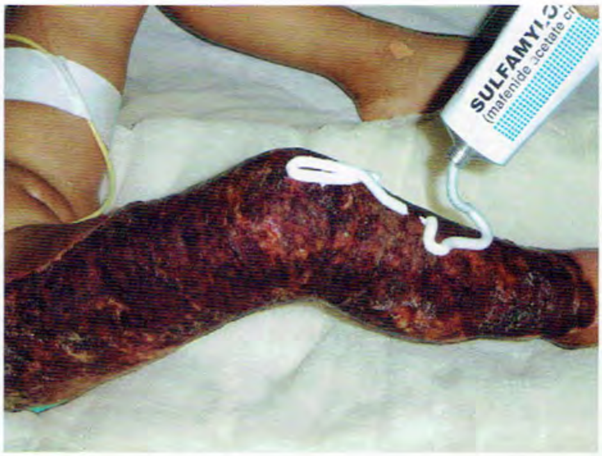

Fig. 20.12F: Patient managed with change of local antimicrobial from SSD to mafenide and third line of systemic antibiotic—Carbapenems with aminoglycoside

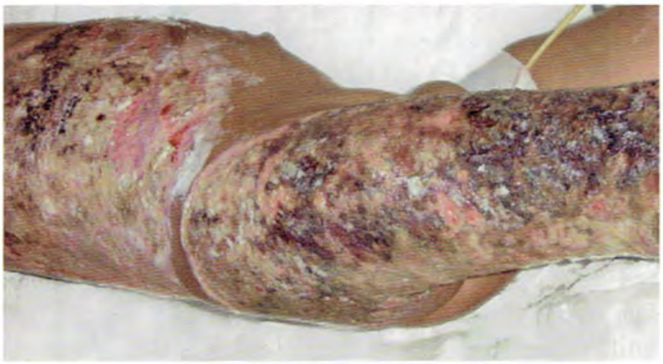

Fig. 20.12G: Improving wounds—Eschar penetrated by mafenide to reach subeschar tissue,tissue edema reduced and general condition improved

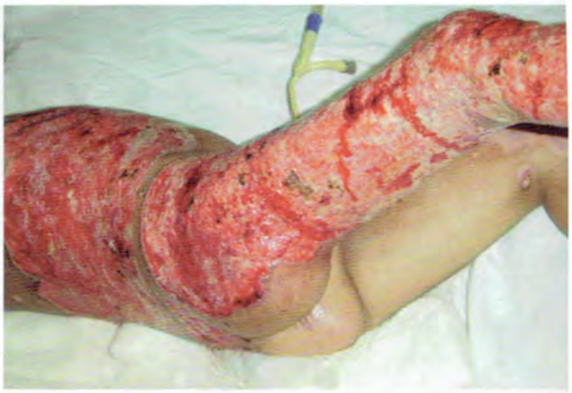

Fig. 20.12J: Healthy granulating wound after 10 days of change in treatment

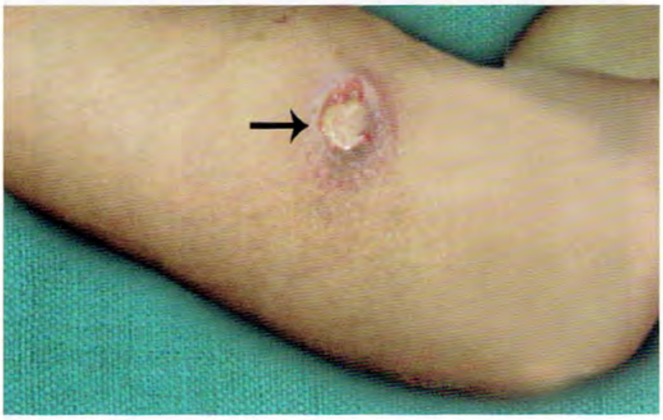

Fig. 20.12H: Ecthyma gangrenosum reduced

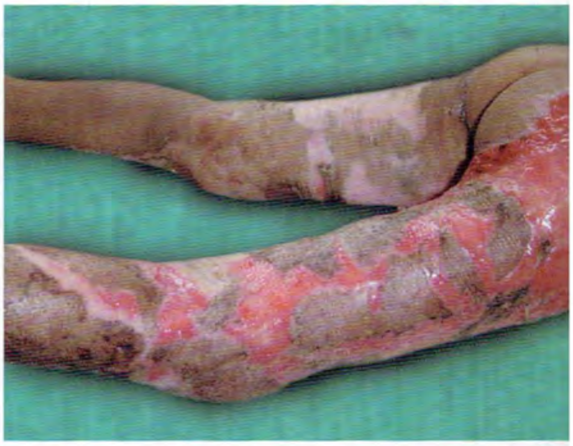

Fig. 20.12K: Same wounds grafted in stages on lower limbs Full graft uptake

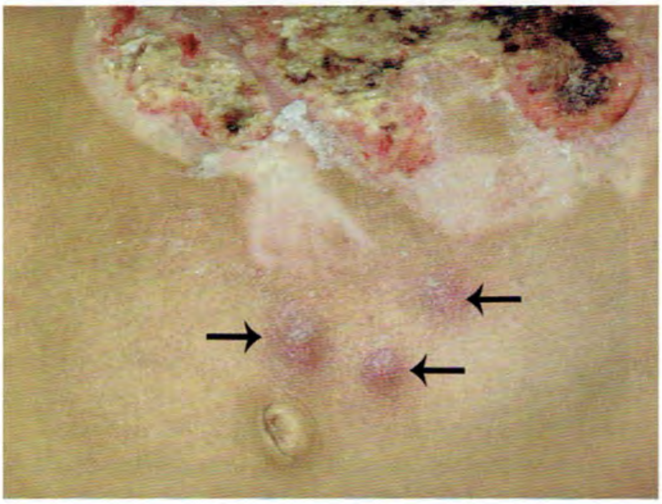

Fig. 20.12I: Satellite nodules drying up

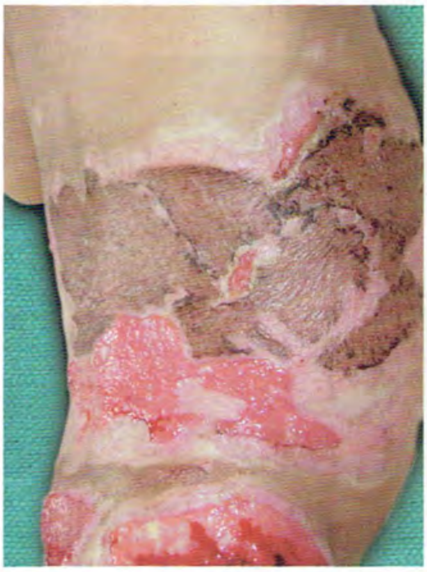

Fig. 20.12L: Successfully grafted back of same patient

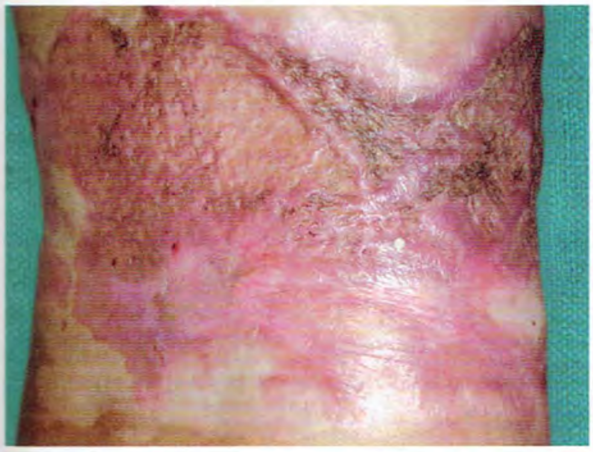

Fig. 20.12M: One month postoperative picture of the back of same patient

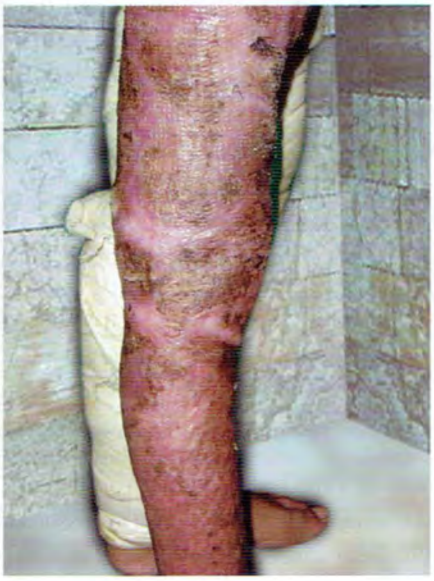

Fig. 20.12N: Legs of the same patient

over to 11.1% mafenide cream is advised (Figs 20.13A to C). This should be applied twice a day. If MRSA (methicillin resistant *Staph. aureus*) is the causative agent, silver ion is more effective and a thick coating of SSD is to be applied and dressing needs to be changed every 8 hours.

Systemic Antibiotics

In stage IIc, hematogenous and lymphatic dissemination demands systemic antibiotic therapy. Ideally systemic antibiotics should have been started much earlier so as to prevent progression to this stage which may ultimately lead to multi-organ failure.

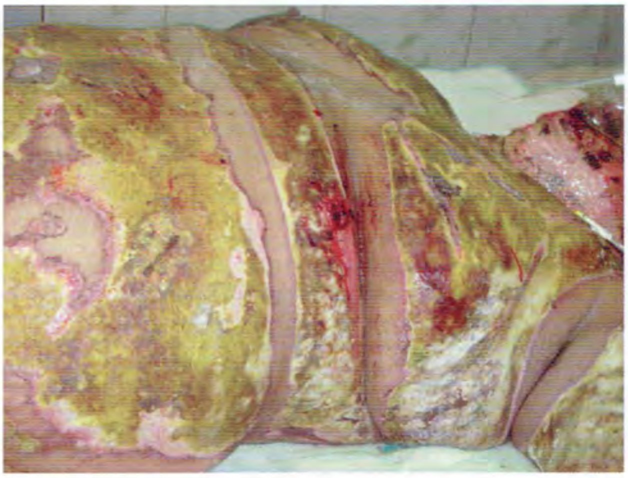

Fig. 20.13A: Superficial burns converted to deep burns by *Pseudomonas* infection which was controlled timely by systemic antibiotics and change of local antimicrobial agent

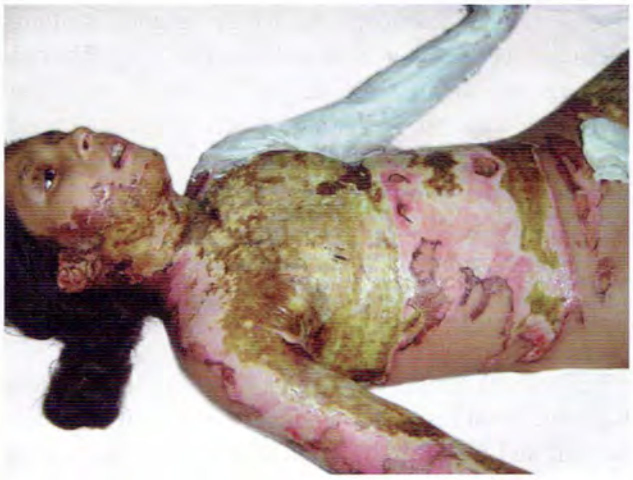

Fig. 20.13B: Same patient with wounds showing improvement

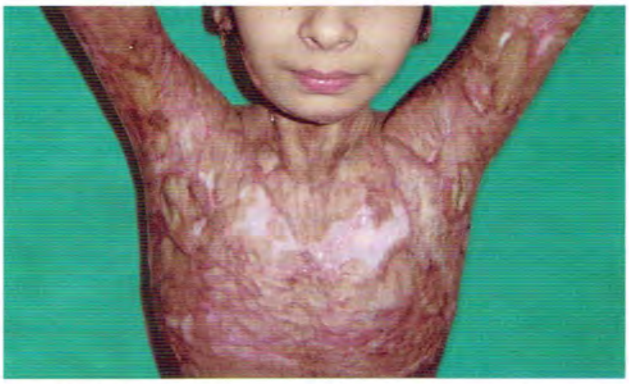

Fig. 20.13C: Finally grafted wounds of same patient–2 months postoperative picture

Appropriate systemic antibiotics should be started depending on the culture sensitivity report. If however, the sensitivity report is not available, empirical antibiotic therapy has to be started as per the ward antibiogram. Every burn unit should formulate its own antibiotic policy according to which the treatment can be started. In the authors' unit, it is a standard practice to use Cefoperazone + Sulbactam or Piperacillin + Tazobactam and Amikacin for beta lactamase producing *Pseudomonas* and *Klebsiella* and *vancomycin* for MRSA. Extended spectrum beta lactamases (metalloenzyme) producing *Pseudomonas* and *Klebsiella* require second generation Carbapenems i.e. Imipenem + Cilastin or Meropenem along with Aminoglycosides.

It is important to diagnose the type of fungal infection accurately because no antifungal provides optimal coverage for all fungi. Because of this varying spectrum of activity of each antifungal agent it is important to identify the infecting fungi accurately, often down to the species level. Intravenous Amphotericin B still has the broadest antifungal spectrum especially *Candida* and *Aspergillus* sp. The older azole agents, fluconazole and Itraconazole cover most *Candida* species but have limited action against moulds. The newer broad spectrum azole, voriconazole has improved mould coverage as well as *Fusarium* and *P. boydii* but no action against *Zygomycetes*. The newer agents like Caspofungin, micafungin and Echinocandin have good action against *Candida* and *Aspergillus* but do not cover *Zygomycetes*, moulds and *Cryptococcus*.

Antimicrobial resistance—There is a worldwide emergence of antimicrobial resistance amongst the majority of human bacterial and fungal burn wound pathogens which makes the therapeutic options limited for effective treatment of burn wound infection.[27,28] MRSA and Methicillin resistant coagulase negative *Staphylococcus*, vancomycin resistant enterococci and multiple resistant gram-negative bacteria which have several types of beta lactamases including ESBL, amp-C beta lactamases and metallobeta lactamases are emerging as serious pathogens in hospitalized burn patients.[27-29] The risk factors for acquisition of antibiotic–resistant organisms include treatment with antibiotics prior to the development of infection, prolonged hospital stay, previous hospitalization, invasive procedures and advancing age. The antibiotic utilization should be rotated or changed on a regular basis as per the changing antibiotic resistance and sensitivity patterns.

Even fungi especially *Candida* sp. has become an important opportunistic pathogen due to widespread inappropriate use of broad spectrum topical and antibacterial agents in burn patients and are now increasing degree of antifungal drug resistance.[30,

Strict infection control practices and app empirical antimicrobial therapy is essential for incidence of infection by these resistant organism

The use of subeschar clysis[32] with a broad s antipseudomonal agent has also been recommend no. 20 spinal needle (half of the dose mixed in saline) to fight the infection locally. This can be just before excision to reduce intraoperative dissen of viable bacteria and toxins.

Burn Unit Antibiogram

All burn units should routinely determine the specific of burn wound microbial colonization and the cha the microbial flora with time and the susceptibility of the organisms. This information can be obtai periodic sampling of burn wounds and other anatom and with a proper liaison between the treating clinici the microbiologist, a yearly antibiogram data can be an periodically. This can then help in selecting and admini appropriate empirical systemic antibiotic, at the sli sign of sepsis, even before the microbiological cultu sensitivity report is available. This saves preciou required to control invasive infection.

Surgical Treatment of Infected Burn Wound

The excision of infected wound should be done general anesthesia, with a scalpel and all tissues above fascia should be excised. In case there is involveme fascia or subfascial tissues, they also need to be excis it is definite that all the infected tissue has been excise resulting wounds should be split skin grafted or co with skin substitute or biological dressing. However, if is any doubt regarding the depth of excision, the wo should be dressed with gauze soaked in 5% mafeni 0.5% silver nitrate solution. The wounds shoul reexamined 24-48 hours later for further excision.

Fungal infection (Figs 20.14A to E): *Candida* infected wounds do not require surgical excision as they rarely in healthy tissue. For these cases dressing with to antifungal agent such as Clotrimazole cream is us effective. If infection is not controlled with the a treatment and involves more tissues locally, excision

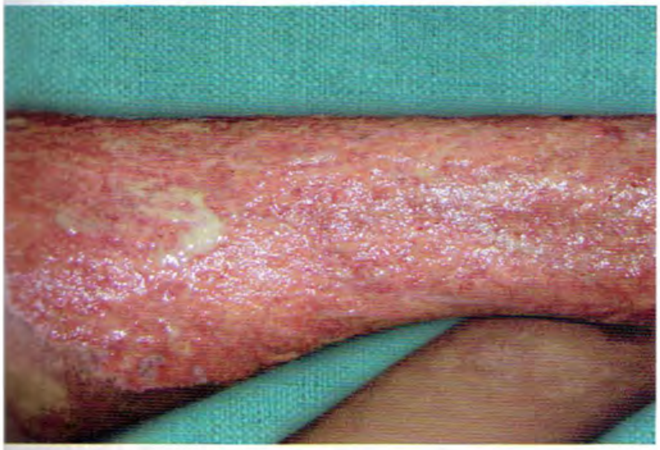

Fig. 20.14A: Fungal infected wounds on lower limb—Patient started on systemic antifungal agents and local clotrimazole cream dressing

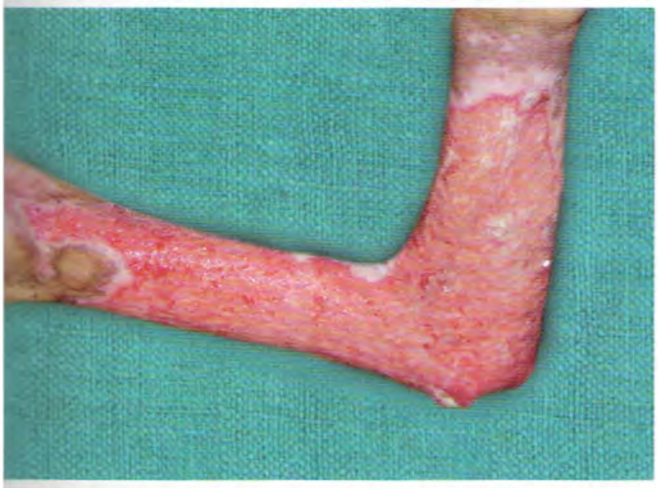

Fig. 20.14B: Same patient's wounds on upper limb

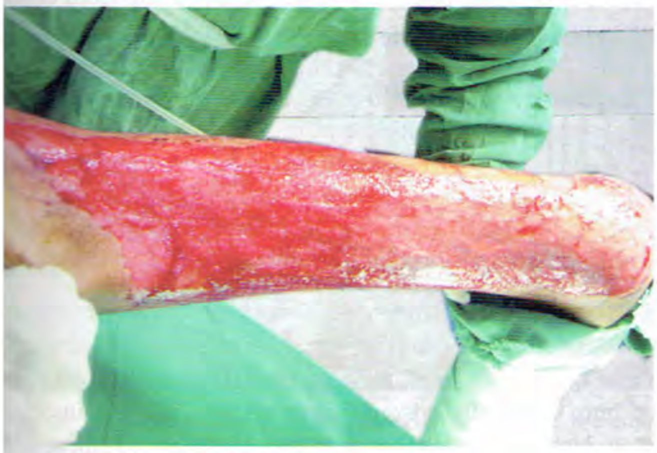

Fig. 20.14C: The wounds improved with local dressing with systemic treatment and prepared for grafting

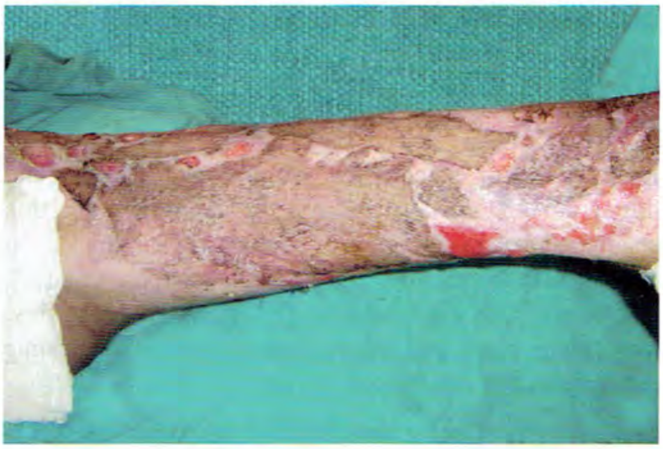

Fig. 20.14D: Raw areas successfully grafted on lower limb -2 weeks postoperative picture

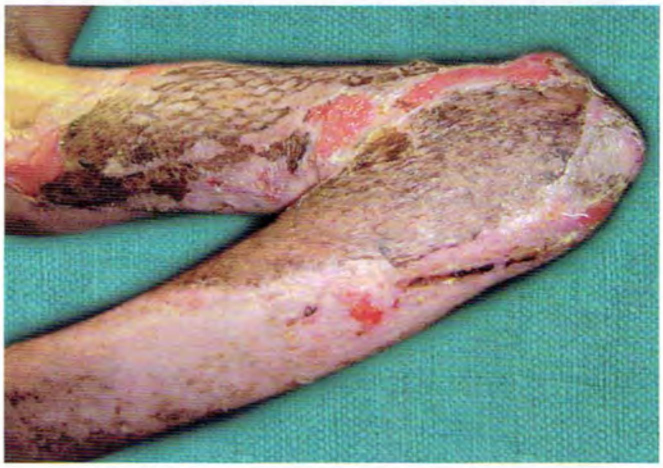

Fig. 20.14E: Same patients upper limb healed with skin graft in two weeks

be necessary. Invasion of the wound is usually seen in filamentous fungi as previously stated, the most aggressive being *Phycomycetes* followed by *Aspergillus* sp. and merits a complete excision of the infected wound followed by twice daily application of topical Clotrimazole cream. Extensive involvement or systemic invasion requires systemic Amphotericin B or other antifungal agents as per sensitivity pattern. In some of these cases if muscles are also involved, an amputation of the limbs may be necessary.

Viral infection[26]: If a patient has unexplained hypotension or other signs of systemic sepsis with no other source and rapidly spreading cutaneous herpetic lesions, the possibility of systemic herpes simplex virus should be entertained and Acyclovir therapy should be instituted.

BURN WOUND IMPETIGO

It is characterized by multiple small superficial abscesses, the causative organism being *Staphylococcus*. If not controlled in time, it can cause extensive destruction of grafted areas or even healed partial thickness burns and donor areas (Figs 20.15A and B). Treatment for this condition involves de-roofing of all abscesses and application of topical antibacterial ointments like Mupirocin (T-bact®, Bactroban®) or Fusidic acid (Fucidin®) twice a day. Vancomycin is required if there is a disproportionate systemic response to this infection.

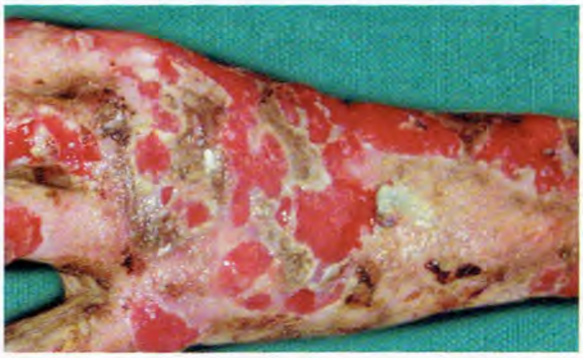

Fig. 20.15A: Staphylococcal infection postgrafting—Typical eaten up appearance of graft applied on hands and arm

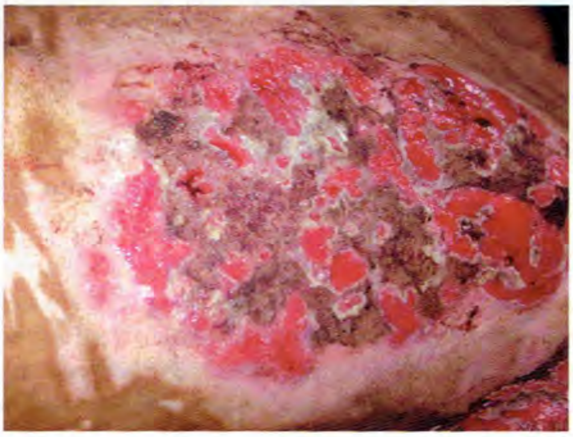

Fig. 20.15B: Same patients grafted chest showing graft eaten up by *Staphylococcus*—Required change of local antimicrobial agent to mupirocin and systemic antibiotics with antistaphylococcal action

SUPPURATIVE THROMBOPHLEBITIS (FIGS 20.16 AND 20.17)

Very often, thrombophlebitis is a cause for septicemia in burn patients as these patients require intravenous (I/V) fluids and frequent parenteral hyper alimentation fluids for a long time with an impairment of host defenses due to burn The incidence of catheter associated infections ha found to be inversely proportional to the distance of th insertion site from the burn wound. Catheter infecti burn patients usually arise from the adherence and mig of burn wound flora microorganisms along the ca surface to the tip.[6,33] Though peripheral and central v catheters should ideally be inserted through normal this may not be possible in case of extensive burns. At t it may be necessary to cut down into a vein throug burn wound itself if other veins have been exhauste patient with extensive burns. All veins which have cannulated have to be examined. Usually there is a red s seen along the length of infected vein with tende indicating thrombophlebitis. If the vein is incised and is a brisk flow of blood from proximal segment this cond can be excluded as the cause for sepsis. But if there is in the vein, culture of the pus should be taken and the e involved vein till the point where it is grossly normal sh be excised completely. Systemic antibiotics are also esse at the same time. To avoid this condition, the I/V site sh be changed every 3 days.

TETANUS IN BURNS

Despite the widespread use of tetanus toxoid which is date the most effective vaccine, tetanus is still a proble most developing countries. The actual incidence of teta associated with burn injury is not reported in Indian literatu although American literature has quoted an incidence of 6.6%.[34,35] Since tetanus prophylaxis is an integral part management of all patients with burns, the incidence tetanus in patients admitted with thermal burns is very ra In the author's unit, tetanus is noticed mostly in elect burn patients and patients who are referred late from oth units not receiving proper tetanus prophylaxis.

In addition to tetanus, gas gangrene and mixed clostridi infections are also seen in burn patients especially with fu thickness burns or electric burns with extensive musc necrosis which is a suitable environment for anaerobi organisms.

Tetanus Prophylaxis

For burn patients who have history of being previousl immunized by basic immunization series plus a booster dos 0.5 ml of tetanus toxoid (TT) given I/M at initial treatme of burn is usually adequate prophylaxis. In case, interv from last booster dose is more than 10 years or if there is delay from time of burning to treatment, 500 units of Huma

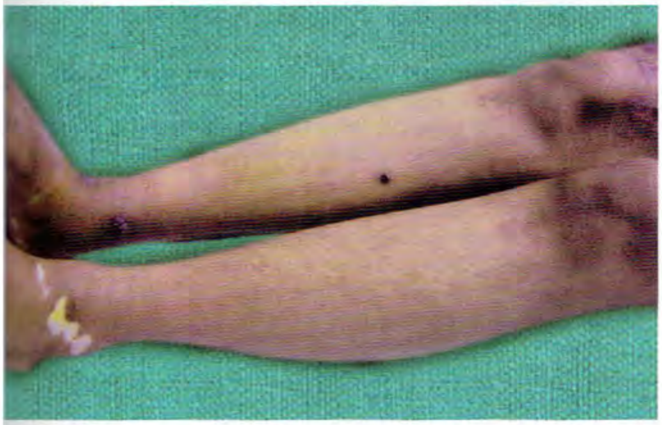

Fig. 20.16A: Deep vein thrombosis in a patient on prolonged central venous line in left lower limb showing gross edema of lower limb and color Doppler showed thrombous extending in popliteal V, femoral vein till external iliac V

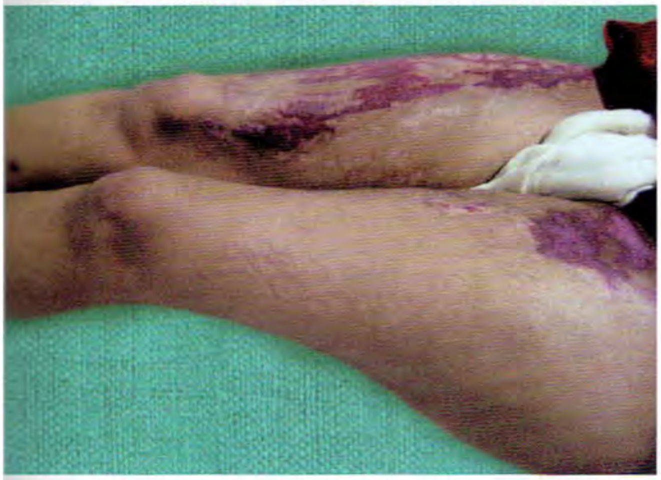

Fig. 20.16B: Grossly edematous left thigh of same patient

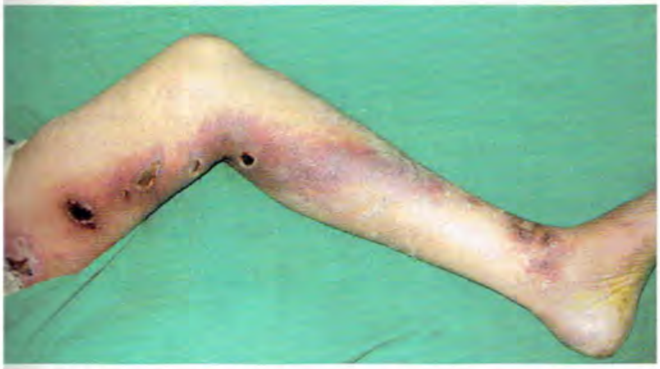

Fig. 20.17: Septic thrombophlebitis all along the length of the great saphenous vein with abscesses presenting superficially at multiple sites with overlying skin necrosis—Required drainage of pus with excision of the entire length of vein

tetanus globulin should be given I/M in addition to tet toxoid with a different syringe at a different site. Pati who have not received basic immunization as per sche should receive initial 0.5 ml tetanus toxoid at time of b followed by 2 injections of 0.5 ml TT at 4 weeks inte and then a booster dose at 6 months to 1 year after the injection. In addition to this schedule, 500 units of hu tetanus immunoglobulin should be also given I/M wi different syringe at a different site.

Tetanus organisms are sensitive *in vitro* to penic and tetracycline. Earlier when the incidence of tetanus to be high, routine antibiotic prophylaxis for tetanus given in the form of crystalline penicillin for the first 7 c following burns. This was, supposedly, to give protec against tetanus till active immunity by tetanus toxoid ta over which is usually from 7-10 days. Topical ther with mafenide also gives protection against Clostri infection.

Treatment of Tetanus in Burns

There are 3 stages of tetanus–first stage is the 'focus infection' stage, wherein there is deposition of Clostri tetani in the burn wound. This infection is well localize the organism does not have strong invasive powers. Dur this stage only local wound care is sufficient, failing wh the second stage sets in. This is the stage where the organi releases highly diffusible neurotoxin into the body flui Treatment during this stage is by neutralization of the to with tetanus antitoxin which may be present in the b from active immunization or should be administe passively. If this stage is not controlled in time, the th stage or neurological stage sets in wherein the toxin fixe the neuromotor cells in the central nervous system. At t stage the antitoxin has no effect on the tetanus neuroto

The neurological or clinical tetanus is diagnosed clinical signs and symptoms and can be mild, severe fulminating depending upon the amount of toxin combi with the nervous system cells. Tetanus typically begins w increase in tone of various muscle groups especially spa of masseter muscle leading to difficulty in opening mo or clenching of teeth with a fixed smile called ' ri sardonicus'. The patient is irritable and complains of inabi to swallow, i.e. dysphagia. As the disease progresses th will be stiffness of neck, back and abdominal muscl Violent contractions of muscles or tetanospasms prod opisthotonus, abdominal rigidity and extension of extremi and these can be initiated with the slightest stimulus. Th can even lead to difficulty in respiration which may mani with hypoxia, cyanosis and even sudden death.

Treatment of established tetanus involves the following steps:

1 Neutralization of circulating toxin by administering 3000 units (1500-10,000 U) of human tetanus immunoglobulin once only at different sites deep intramuscular.
2. Removal of source of toxin, by excision of presumed site of entry which is the burn wound, is possible only in small burns. Excision of major burns for treating tetanus is not recommended.
3. Antibiotics in the form of Penicillin or Tetracycline also help in eliminating tetanus organisms.
4. Sedation to control tetanic spasms in the form of diazepam is useful though toxic doses should be avoided.
5. Maintenance of respiration – All patients with tetanus should undergo tracheostomy and ventilator should be kept ready, if needed.
6. Supporting care: Fluid electrolyte balance, renal function, ventilation, nutrition and prophylaxis for pulmonary and urinary tract infections have to be looked into as well.

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4 Section

Burns of Special Types

Chapter 21

Electrical Burns

SP Bajaj

INTRODUCTION

Electricity is known to mankind since time immemorial. Thales of Miletus in 6th century BC wrote that rubbing fur on amber would cause attraction between the two substances. This is probably what we understand as static electricity today. The word 'electricity' is derived from Latin "electricus", meaning amber-like.

Luigi Galvani (1781) demonstrated that electricity was the medium by which nerve cells pass signals. In 1821, Michael Faraday invented the electric motor. Commercial use of electricity became possible only in mid-eighteenth century. Electricity is probably the most dangerous commodity which we use everyday and in every moment of our lives. Development of electricity drastically affected social thinking. During the age of mechanization, there was an emphasis on splitting the work into many sections and then creating a sequence of completing each section separately. With the development of electricity there was emphasis on work being done in "totality, simultaneously and instantly."

EPIDEMIOLOGY

With the widespread use of electricity its dangers also started showing its effects. First recorded death from an electric current was in Lyons, France in 1879, where electricity was used to lighten a stage for opera. In US, Samuel Smith was the first person who died from electrocution from a generator in 1881 in Buffalo, New York.[1]

With the continued growth of the electrical industry, there was a simultaneous rise in number of electrical injuries and deaths. Today in US 0.8 to 1% accidental deaths[2] are caused by electricity. Electricity causes around 1000 deaths each year in industrial workers.[3] Between 1992 and 1998, 2287 people died from electricity at their place of work. 32807 people sustained different degrees of burns and burn shock, keeping them away from work. In US, roughly one person dies everyday at his place of work by an electrical injury and over 13 days of work are lost per electrical injury. No such detailed data is available from Europe, Indian subcontinent, Africa or Asia.

All over the world, electrical injuries are the second most important cause for admission to a burns unit. The percentage varies from 6% in US to 20% at many Asian and African burns units. 40% of these patient's with serious electrical injuries die.[4,5] There are two age groups which are frequently involved in electrical injuries. The first is that of children less than 6 years age who have a high incidence of oral electrical burns because of oral contact with electric cords or when they come in contact with electric poles or wires while playing. The incidence decreases in teenage years and then increases in adults as they start working.[6,7] Approximately one third of electrical injuries involve electrical workers and one third affect construction workers. Male to female ratio in childhood electrical injuries ranges from 1:1 to 2:1 with male predominance. Amongst adults, 93% of the victims are men.

Electrical burns are often regarded as equivalent to thermal burns, as electrical energy is converted to thermal energy when it comes in contact with the body. However, many effects of electricity on the human body are other than that of heat. For its complete understanding, some basic knowledge of electrical physics is essential.

PHYSICS OF ELECTRICITY[8,9]

Electric Current

Electron, Proton and Charge: Movement of electric charge is known as electric current. Charge on electron and proton is same and is expressed as negative or positive. This concept of negative and positive charge was given by Benjamin Franklin. Symbol "Q" is used to express the amount of charge and is expressed in Coulombs. Each electron or proton has a charge of 1.602 × 10^{-19} coulombs. Electron is negatively charged while proton is positively charged.

Unit of Current

Ampere: Passage of electrons at a given point in one second is expressed in amperes. When 6.242 × 10^{15} electrons are passed from a given point in one second, it is called passing of 1 ampere of current.

Voltage

In all conductors electron move in a random fashion until driving forces (electromotive force) are applied to them. This is expressed in Volts. A large volt exerts a large force which makes electron to move in a short period thus more current to flow in a short period. Thus, in all transmission cables high voltage is used. More the voltage less is the safety to the human body. High voltage is also used to move electric train and underground metro. In UK and some European countries this is 11,000 volts. In India, it is 25,000 volts. House-hold current in American sub-continent is supplied at 110-120 volts. In Indian sub-continent, Europe, Australia and most of Africa it is 210-220 volts.

Resistance

All substances have some resistance to the flow of current. Substances which have low resistance are called good conductors while substances with high resistance are called bad conductors. In a substance which is a bad conductor electrons are fixed and have hardly any movement while in a good conductor electrons are loosely placed. Substances which have no resistance are called super conductors. Human skin provides high resistance which may be as high as one million ohm/cm^2. Resistance in human skin decreases in summer because of sweating. It is less whenever it is wet. Resistance is also low in children and at extremes of age because of thinness of skin epidermis. In the skin, dermis has lesser resistance as compared to epidermis. Resistance is also related to the circumference. Thus, chest and abdomen with greater circumferences provide lesser resistance while a finger with lesser circumference greater resistance. Similarly, current flow in the larg with bigger circumference provides less resistan smaller vessels provide more resistance and hence likely to get thrombosed.

Further, current density increases in smaller vesse it is reduced in larger structures. Current density ha effect in the human body. With arteries having rapidly blood, current density at any given point and at an time becomes less because of flow of blood. While density is more in the veins because of slower flow o Therefore, thrombosis and stoppage of blood flow in veins and comparatively less in arteries.

Ohm's Law

There is a definite relationship between flow of (ampere), force of current flow (voltage) and resist flow (ohm). This relationship was best described b and is called Ohm's law. Ohm's law states

Current (I) = *Voltage* (E)/*Resistance* (R)

It means current is directly proportional to volta inversely proportional to resistance, more the voltag the current, more the resistance less is the flow of c

Joule's Law

Further, with greater resistance, greater numbers of ele collides with each other and generate more heat. Th described by Joule and is explained mathematically by J Law.

Power (P) = Voltage (E) × Current (I)
Further E = I × R
Therefore P = I^2 × R

Heat generated in a current source due to flow of c is the indicator of power. Watt is the unit of electrical 1 ampere of current flow through 1 Ohm of resistanc second is one Watt of energy generated. One Watt per s produces 0.24 calories of heat.

Type of Current

Direct current (DC) or Alternate Current (AC).

There were great discussion about safety and c current supply between DC (invented by Edison) a (invented by Tesla) in 19th and early 20th centuri present, Alternate Current (AC) has entirely supe Direct Current (DC). It is cheaper and easy to tran into any desired voltage. Direct Current is restricted to the chemical and metallurgical industry. It is also used in the automobiles. However, it is believed that AC current is more dangerous to life as compared to DC current. There is no 'let go' effect in AC current while in DC current a person is thrown out after a short muscle contraction. AC current flow regularly reverses its direction and thus creates a sign wave. Direct current flows in a constant direction. All batteries deliver Direct Current. Use of UPS and alternators are based on Direct Current.

Electric Arc[10,11]

An electric arc is formed between the two substances of high potential difference. In humans, it is high voltage power source and grounded body of the man. Arc is made up of ionized particles which are driven by high electromagnetic force (Voltage) between the source and human body with air space in between. Temperature of the ionized particles may reach as high as 3-4 thousand degree Celsius. Such high temperatures can melt any metal. Arc is seen around for a distance of many centimeters even in case of few thousands volts.

Burn occurs at the point where arc strikes the human body. Electric arc remains an important cause in high voltage electrical injuries.

EFFECT OF CURRENT SUPPLY ON HUMAN BODY (FLOW CHART 21.1)

Effects of current on human body are well described. However, the mechanism(s) of action is (are) not equally well understood. Most often, this effect is described on the basis of conversion of electrical energy into the heat energy. Therefore, it is justifiable to think about the resistance offered by the body to the flow of current. Different body tissues provide different resistance to the flow of current. Maximum resistance is provided by the deepest structure i.e. bones and the most superficial structure i.e. epidermis of skin. However, there is an alternative view also which says that human body works like a volume conductor and there is no effect of resistance of individual tissue. The latter part appears to be correct. Because of bigger volume of chest and abdomen lesser damage is seen to these structures as the heat dissipates over a larger area. Because of smaller volume of fingers and limbs more damage is seen at these points[12] (Fig. 21.1).

In humans and in the animal kingdom as a whole, the mechanical energy is converted to chemical energy which is converted into electrical energy. All final commands in the body tissues and cells are by electrical signals only.

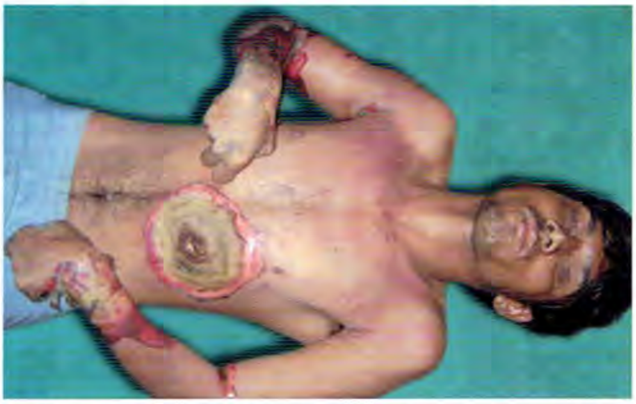


Fig. 21.1: Electrical contact burn in a young patient who was standing next to a wall and holding a pole with both his hands which accidentally touched a high tension wire causing severe burn in both the hands–Gangrene of both hands and exit wound in lower chest and abdomen which is less than the limb injury because of the larger circumference of the chest

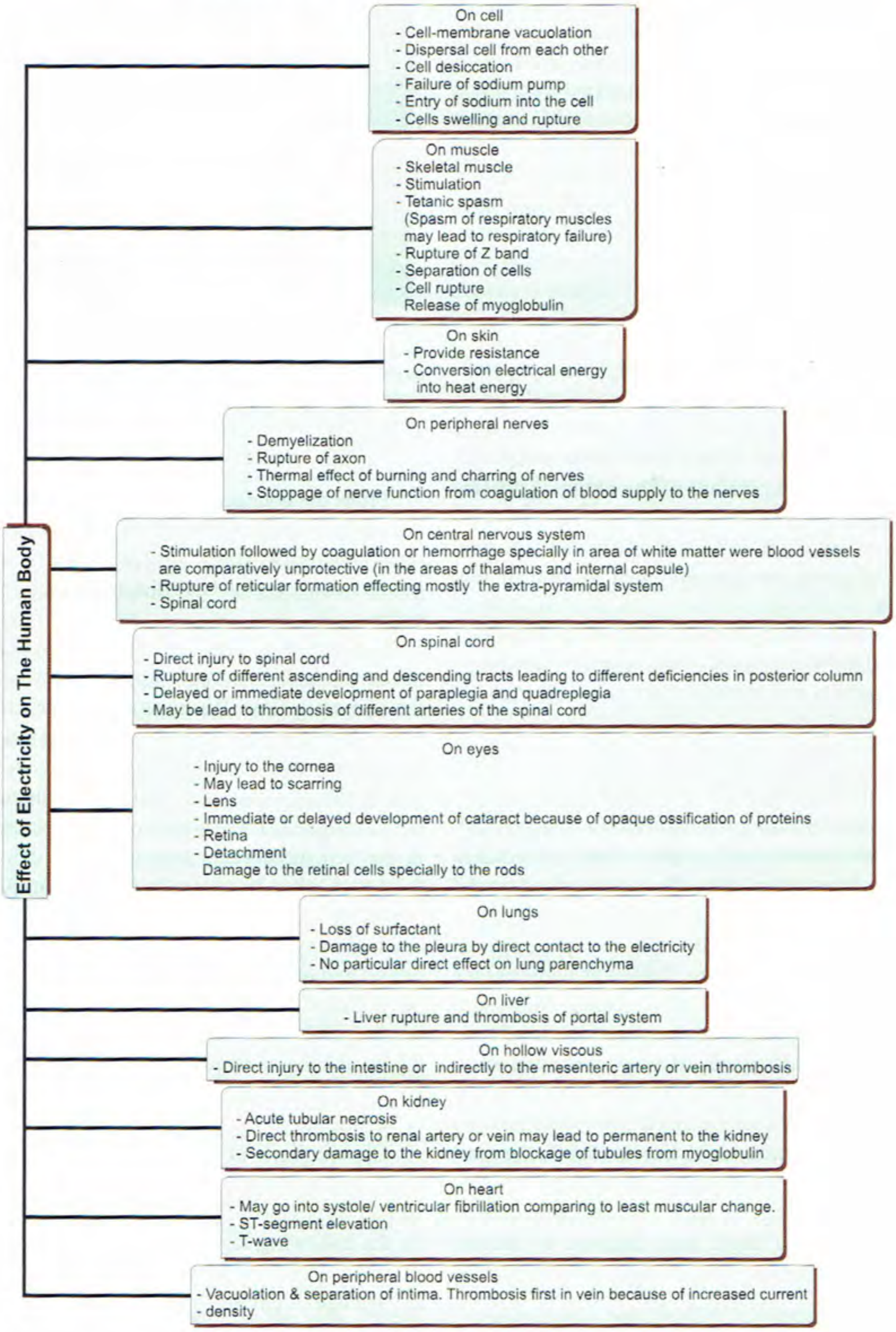
From the conduction point of view best conductors are nerves followed by blood vessels and muscle. Tendon, tendon-sheath, deep fascia, dermis, epidermis and subcutaneous tissue have the resistance in this ascending order.

Unfortunately not much has been explained about how the current affects the cells and tissues which are permitting the current to flow. We must remember while gravitational forces keep the cells and masses pulled towards each other, electromagnetic force is 10^{42} times stronger than the gravitational force and pushes the electron apart. This electromagnetic force is responsible for creating vacuolation in the cell membrane, separating the cells and detaching from each other, blocking the sodium pump, leading to entry of sodium into the cell, stopping the cell function and finally death. At any given time, cell may remain in a hibernation period and once the effect of current has gone may recover fully. Unfortunately, these effects are not fully understood and its mechanism remains debatable. Effect on brain and the heart muscle show considerable period of hibernation hence often complete recovery after initial sign of infarct or hemorrhage.

Classic work of Kouwenhoven (1949)[13] provides the basic understanding about the effect of electricity on the human body. According to him effects of the current depends on the following factors.

1. Type of current used i.e. AC or DC
2. Voltage of circuit
3. Resistance
4. Amperage or amount of current.
5. Pathway of current
6. Duration of contact.

Flow chart 21.1: Effect of electrical current of human body



Effect of Electricity on The Human Body

On cell
- Cell-membrane vacuolation
- Dispersal cell from each other
- Cell desiccation
- Failure of sodium pump
- Entry of sodium into the cell
- Cells swelling and rupture

On muscle
- Skeletal muscle
- Stimulation
- Tetanic spasm (Spasm of respiratory muscles may lead to respiratory failure)
- Rupture of Z band
- Separation of cells
- Cell rupture Release of myoglobulin

On skin
- Provide resistance
- Conversion electrical energy into heat energy

On peripheral nerves
- Demyelization
- Rupture of axon
- Thermal effect of burning and charring of nerves
- Stoppage of nerve function from coagulation of blood supply to the nerves

On central nervous system
- Stimulation followed by coagulation or hemorrhage specially in area of white matter were blood vessels are comparatively unprotective (in the areas of thalamus and internal capsule)
- Rupture of reticular formation effecting mostly the extra-pyramidal system
- Spinal cord

On spinal cord
- Direct injury to spinal cord
- Rupture of different ascending and descending tracts leading to different deficiencies in posterior column
- Delayed or immediate development of paraplegia and quadreplegia
- May be lead to thrombosis of different arteries of the spinal cord

On eyes
- Injury to the cornea
- May lead to scarring
- Lens
- Immediate or delayed development of cataract because of opaque ossification of proteins
- Retina
- Detachment Damage to the retinal cells specially to the rods

On lungs
- Loss of surfactant
- Damage to the pleura by direct contact to the electricity
- No particular direct effect on lung parenchyma

On liver
- Liver rupture and thrombosis of portal system

On hollow viscous
- Direct injury to the intestine or indirectly to the mesenteric artery or vein thrombosis

On kidney
- Acute tubular necrosis
- Direct thrombosis to renal artery or vein may lead to permanent to the kidney
- Secondary damage to the kidney from blockage of tubules from myoglobulin

On heart
- May go into systole/ ventricular fibrillation comparing to least muscular change.
- ST-segment elevation
- T-wave

On peripheral blood vessels
- Vacuolation & separation of intima. Thrombosis first in vein because of increased current
- density

To this a seventh factor can be added and that is area of contact. He also confirmed that the main mechanism of immediate death from electricity was by induction of ventricular fibrillation.

Type of current causes significantly different effects on the human body. Skin offers greater resistance to the Direct Current than to Alternate Current. 3-4 times more Direct Current is required in producing the same effect as alternate current. Direct Current does not produce muscle contraction as produced by the Alternate Current. With increasing alternating current the magnitude of muscles contraction keeps on increasing and subjects hold on the contact point becomes so intense that he/she cannot release the conductor. Maximum current a person can hold and still release on one's will is called "Let go" current. Such strong muscle contraction may lead to fractures and/or dislocation of joints. Threshold for 'let go' effect in both sexes is different, probably because of different muscle strength. It is 15.87 mA in men and 10.5 mA in women with normal commercial supply of 60 cycles of alternating current.

In contrast to low voltage, high voltage direct current is more lethal than alternating current (Flow chart 21.2). Alternating current of 60 cycles with as low voltage as 60-65 volts has resulted in fatal accidents. However, the threshold of 24 volts has been described as safe. Lower volts than this in any circuit have been found to have no lethal effects on the human body.

Actually it is the total current entering the human body which is of concern. All effects are based on the total amount which has entered into a human body. This was best described by Hodgkin BC[14] in 1974. According to him 1 mA of current leads to just tingling or perception which becomes painful when increased to 5 mA. At 60 mA, it leads to cardiac fibrillation, burns at 5000 mA and respiratory arrest at 10,000 mA. A person with high voltage injury develops respiratory arrest and is more likely to revive, compared to low voltage injury where the heart goes into fibrillation and is less likely to be revived.

Pathway(s) of current in the human body is (are) of extreme importance.[14] When heart or brain comes in the path of a current, it is more likely to be lethal. Further, longer the duration of contact, greater are the chances of damage to the vital structures. Thus, a current passing from skull to feet is more lethal than a current passing from right upper limb to the right lower limb (Figs 21.2A to C). Similar current passing from left hand to right hand will have more lethality because of involvement of heart in its path.

From a practical point of view, an electrical injury will have more local effects (burns) when skin provides higher

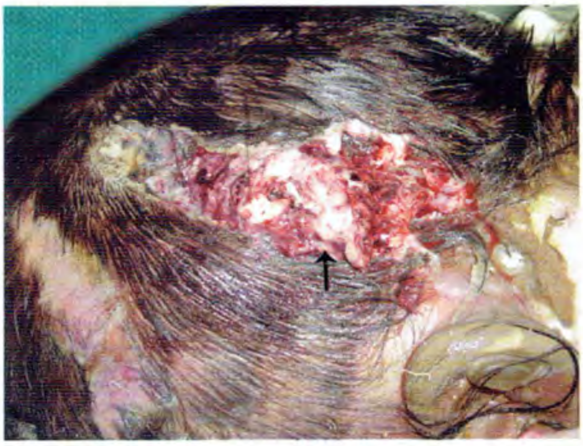


Fig. 21.2A: A 10-year-old child sustained high tension injury due to accidental fall of 11000 V cable falling on her while playing—Entry wound in skull with loss of both tables and brain matter coming out of the wound

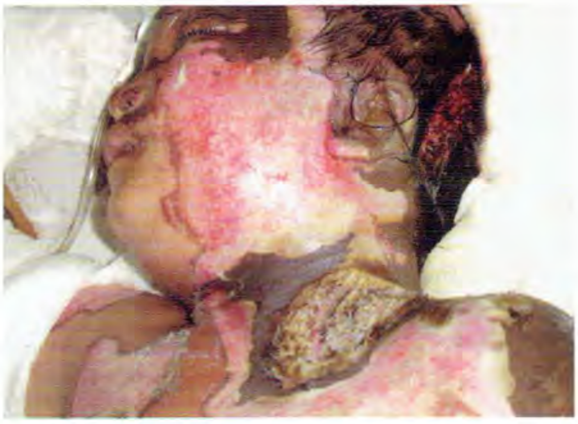


Fig. 21.2B: Another area of contact with wire in the neck causing a deep wound exposing the carotid vessels , with deep flash burns in the surrounding area on face and chest

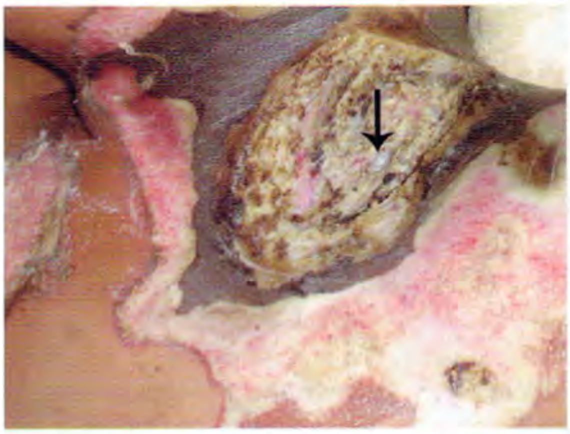
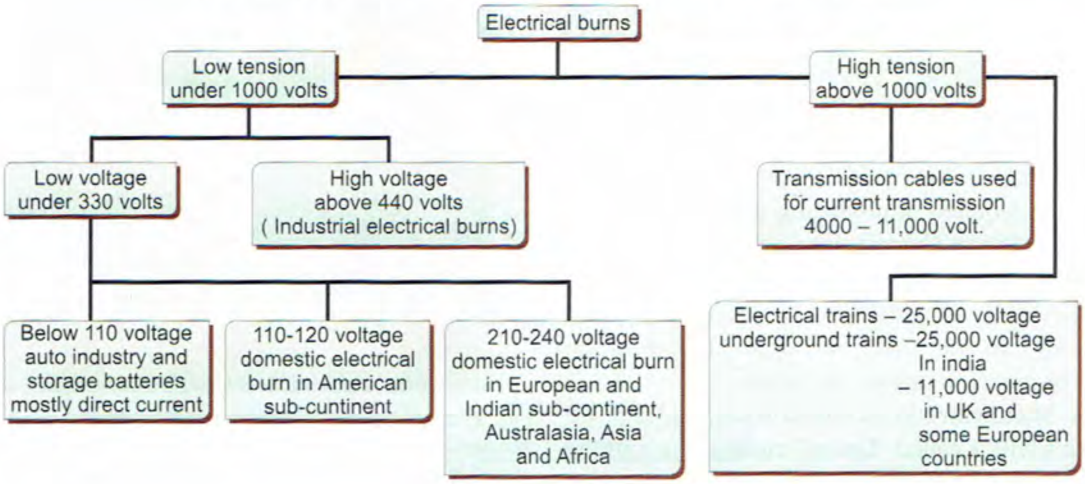


Fig. 21.2C: Third area of contact in same patient below the axilla exposing pleura requiring chest tube drainage for pneumothorax

Flow chart 21.2: Types of electrical contact burns depending on voltage


Electrical burns
Low tension under 1000 volts
High tension above 1000 volts
Low voltage under 330 volts
High voltage above 440 volts (Industrial electrical burns)
Transmission cables used for current transmission 4000 – 11,000 volt.
Below 110 voltage auto industry and storage batteries mostly direct current
110-120 voltage domestic electrical burn in American sub-cuntinent
210-240 voltage domestic electrical burn in European and Indian sub-continent, Australasia, Asia and Africa
Electrical trains – 25,000 voltage underground trains –25,000 voltage In india – 11,000 voltage in UK and some European countries

resistance and will have more severe systemic effects when the skin has lower resistance.

One must understand that during short-circuiting electrons randomly collide with each other producing intense heat and flash. This intense heat and flash leads to burns (Fig. 21.14). This intense heat or flash is for fraction of a second. Contact with human body will produce extensive burns. These are mostly superficial burns. Sometimes clothes catch fire from these intense flashes and produce deeper burns. These burns are truly thermal burns although the causative agent is electricity. In these injuries, there are no systemic or local effects of electric current on the human body.

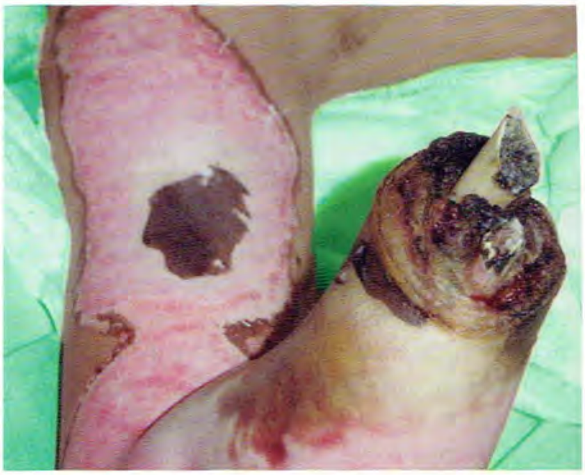


Fig. 21.3: High voltage contact injury causing autoamputation level of forearm because of necrosis till bone

TYPES OF ELECTRICAL INJURIES (FLOW CHART 21.3)

There are five types of presentation seen in electrical injury.

1. True electrical injury: In which body becomes a part of the circuit and injury by the passage of current through the body is manifested by entry and exit wounds. This can be either low voltage (< 1000 V) or High voltage (> 1000V) injury (Figs 21.2 and 21.3).
2. Flash burns: These are surface thermal injuries involving exposed areas of the body like face, forearm and hands. These are sustained by flashes produced during sparking which come in contact with the skin and the heat produced during the process causes burns.The current does not enter the body at all. The temperature in the spark may be as high as 4000°C and though it lasts for a fraction of a second, it can produce full thickness burns (Figs 21.4 and 21.5).

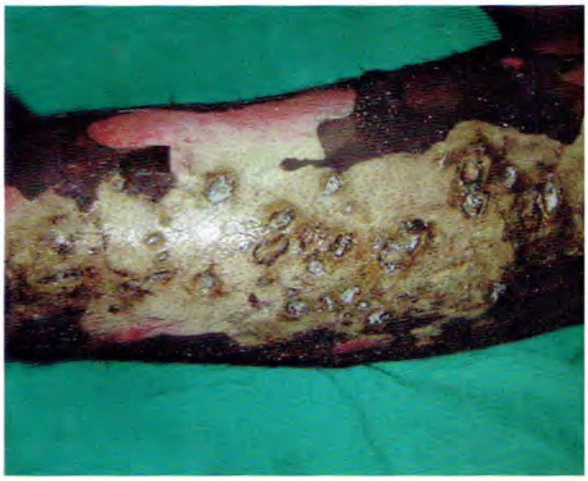
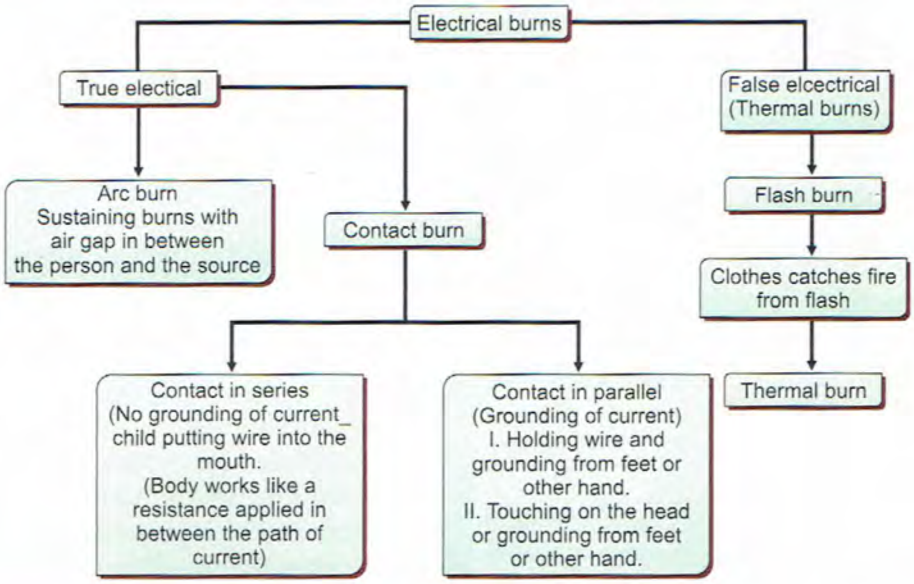


Fig. 21.4: Full thickness flash burns

Flow chart 21.3: Types of electrical injury


Electrical burns
True electical
False elcectrical (Thermal burns)
Arc burn Sustaining burns with air gap in between the person and the source
Contact burn
Flash burn
Clothes catches fire from flash
Contact in series (No grounding of current_ child putting wire into the mouth. (Body works like a resistance applied in between the path of current)
Contact in parallel (Grounding of current) I. Holding wire and grounding from feet or other hand. II. Touching on the head or grounding from feet or other hand.
Thermal burn

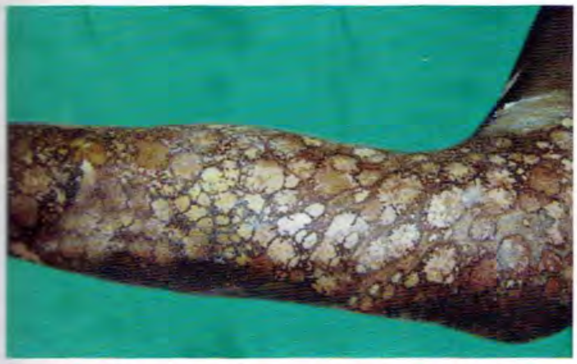


Fig. 21.5: Typical deep dermal injury in flash burns

3. Flame injuries: These are caused by spontaneous combustion of victim's clothes when they come in contact with an electric arc. This is also a pure thermal injury.
4. Lightning:[15,16] Consists of hundreds of million volts lasting from a fraction of a second, often resulting in a flashover phenomenon in which majority of the electrical energy flows over the victim's body rather than through it. The burn due to lightning can vary from minor burns to major burns from ignition of the victim's clothes or heating of the metallic objects on the victim's body like jewellery, coins or dental fillings. The cutaneous burns from lightning are seen as feathery or arborescent, linear or punctate pattern. Respiratory arrest or cardiac arrest with unconsciousness may occur and requires prompt resuscitation with cardiac massage and ventilation.
5. Electric shock or electrocution[9]: It is usually suffered by electricians who describe the feeling as balls of light when the current passes through the body followed by trembling, pallor, sweating and feeling of apathy or euphoria. The patient may become unconscious because of cerebral edema and raised intracranial pressure. Many a times there may be cardiac and respiratory arrest requiring cardiac massage and artificial respiration. There may be temporary deafness or blindness with auditory hallucinations and stiffness or paralysis of limbs. These can last for a few minutes to hours. Altered sensorium may also be temporary.

LOW VOLTAGE INJURY (FIGS 21.6 TO 21.9B)

These injuries usually take place at home. Malfunctioning lawn mower, electric kettle, iron etc. are the causative agents for these injuries. Here the current involved is 110-120 volts (American sub-continent) and 210-240 volts (European, Indian, African and Asian sub-continent). Where three phase electrical current is used as in industries, it may be 440 volts. In all these instances, usually hands are involved and the injury is local. There is practically no systemic effect of electrical current. However, sometimes ECG changes are seen in the form of elevated ST segment/T-wave depression.

Oral Cavity Burns (Figs 21.10A and B)

These injuries are seen in infants and toddlers. An inquisitive child tries to explore the wire by taking it into the mouth

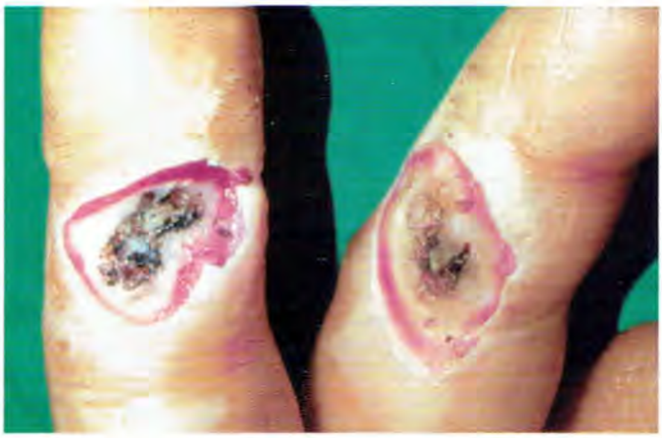


Fig. 21.6. Low voltage electrical burns of fingers treated by primary excision and grafting. No involvement of tendon and nerves

and chewing it with the gum or newly erupting teeth. In majority of these cases both the wires, positive as well as negative, are taken into the child's mouth. Salty watery contents of the mouth allow the current to flow out through the same wire (contact burn in series). Here child's mouth[17] works like a resistance added in the flow of current. The current is converted to heat energy (1000-1500°C) leading to extensive local damage of the oral cavity involving lips, mucous membranes of oral cavity and tongue. An occasional complication is a secondary hemorrhage from labial artery which may occur even many days later when the scab over the artery is dislodged by the child.[18]

In the 2nd type of contact burn (contact in parallel) current passes out from the body to the ground from child's wet diaper, feet or buttocks. This invariably led to serious cardiac problem and may prove fatal (around 5% of the cases).

In most cases, upper lips, lower lips and angle of mouth are involved either alone or in different combinations. Sometime tongue and cheeks are also involved resulting in massive edema. Usually it does not lead to any respiratory problem or any difficulty in swallowing. Excessive salivation and dribbling are seen.

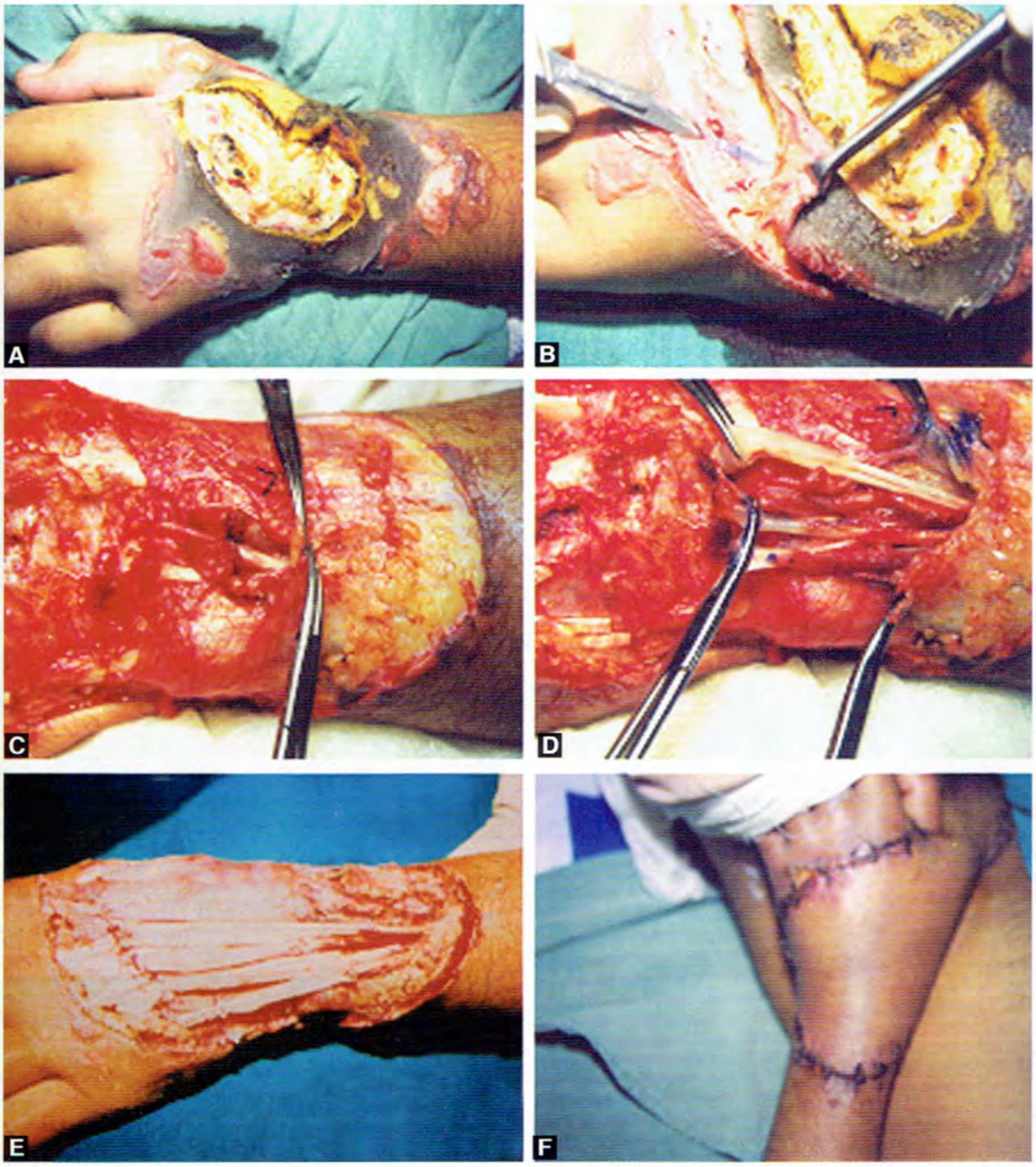
Management

First aid at site of injury: Whenever a victim of electric injury has to be rescued, the safety of the rescuer is of prime consideration. First, the electrical supply should be turned off or the patient should be removed from the electrical source with the help of a well insulated, wooden or rubber coated tool. Next, attention has to be directed towards airway, breathing and circulation of the patient and
steps for basic life support have to be taken. I
desirable that the general public should be conve
this basic first aid knowledge.The patient shou
shifted immediately to the closest hospital. W
minutes of resuscitation, if possible, cardiac mo
the patient is essential because most arrhythn
within the first few minutes to an hour after an
injury. Defibrillation at the scene of injury or a
possible may save a patient's life who is having an
Therefore, all patients must have an ECG done a
be monitored for 48-72 hours for cardiac status. I
more important if there are some changes in the EC

A venous line is kept ready for manageme
drug administration for cardiotropic drugs. U
intravenous fluids may be required for resuscitati
small and localized electric burns. In fingers and ha
assessment must be made of the circulation as i
closed compartments and similarly at wri
compartment may develop because of tight car
These may require urgent release.

After the initial resuscitation patient needs to
to a specialized burn unit where decision regardi
management of the burn wound can be taken. A
are deep in contact burns, excision of burn can be u
once patient appears to be stable from the cardio
point of view. However, person performing these
should be well trained in providing flap cover to t
regions. Sometime at the wrist and dorsum of I
there may be damage to flexor or extensor tendo
be direct injury to the median or ulnar nerve. If in
nerve is seen excision of nerve is done and ner
can be undertaken once one is confident th
successfully excised all the dead tissues. Thi
associated with flap cover, especially areas wh
are excised at the wrist level or carpal and ulnar t
opened (Figs 21.7A to F).

Similarly hand burns are seen in infants an
due to putting the fingers into the electric sockets.
with the development of better and safe childpro
these electric burns in children have become r
Electrical burns in children because of soft pliabil
accumulate lots of fluids and are more likely t
ischemic changes or median and ulnar nerve dam
involve wrist. An early surgery should always be c
in children and one must release the carpal and ul
and also provide a flap cover. In fingers, a cr
flap, flag flap or meta-carpal artery flaps can b
cover the fingers (Figs 21.8 and 21.9).



Figs 21.7A to F: Low voltage electrical injury involving extensor surface of the hand. Showing different levels of involvement with in between normal tissue while deeper necrosis of tendon or thrombosis of veins. Total reconstruction by providing tendon and cover. Cover by pedicle groin flap preferred over free flap

Management of Oral Cavity Burn

All these burns are confined to infants and children hence management should take into consideration, growth and further development. Electrical burns of mouth extend from angle of mouth to the lower lip with injury to the gum, bone and developing teeth. There are two opposing views of thought in the management of mouth burn. One believes in early excision and primary cover. This provides sufficient vascularised tissue for the growth of bones and tooth buds. The other view is that while trying to do the early excision one may excise too much of tissues which may recover on their own. As mouth is a specialized structure, no

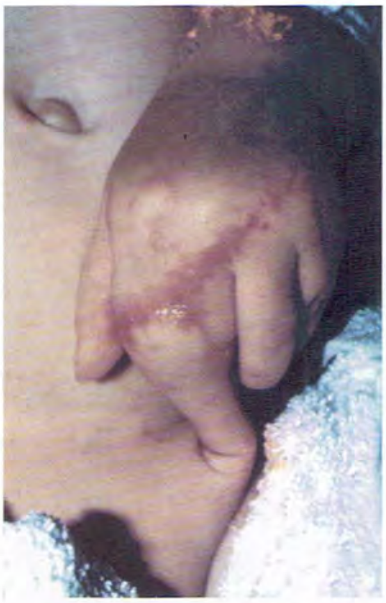


Fig. 21.8: A case of electrical socket injury involving index finger and dorsum of hand. Dorsum primarily excised and closed. Finger covered by pedicle groin flap

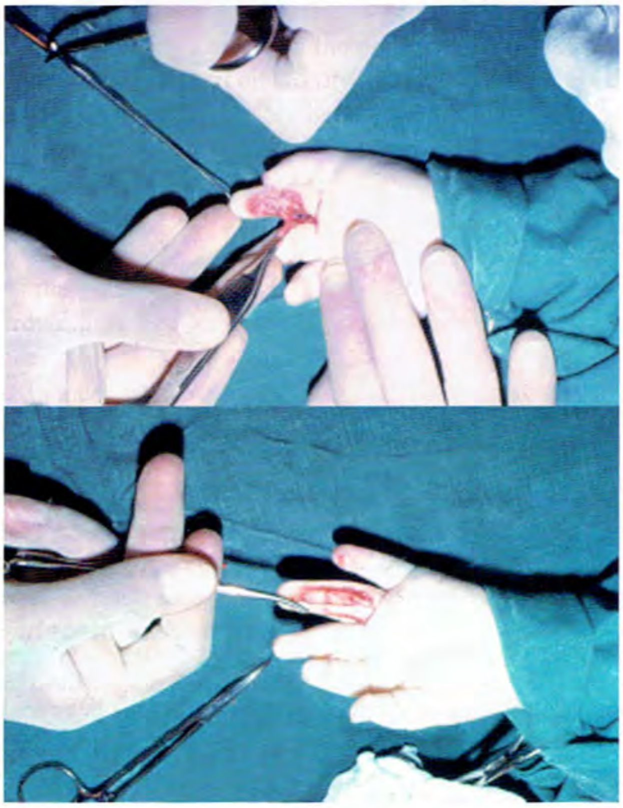


Fig. 21.9A: Domestic electrical burn involving inner aspect of the ring finger. An electrical socket injury. Exposed tendon treated by cross finger flap cover

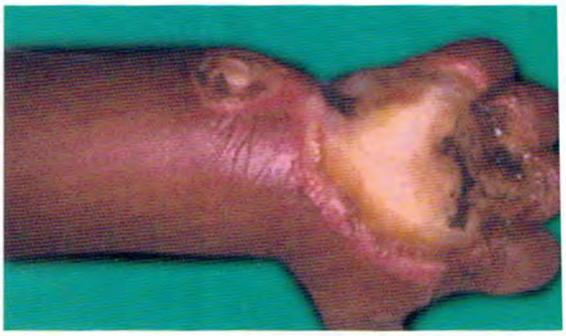


Fig. 21.9B: Electrical burn in a child from domestic electri 220-240 volts with involvement of extensor surface of h wrist

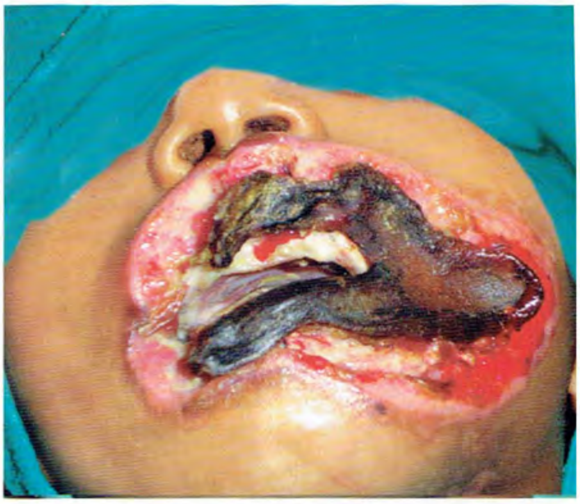


Fig. 21.10A: A 8-year-old with oral cavity burns due to accid of high tension wire over face, leading to necrosis of both u lower lips and left cheek

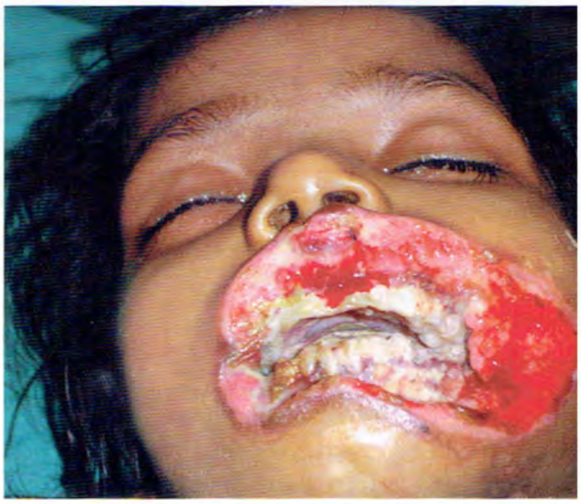


Fig. 21.10B: Defect after removal of eschar exposing both and teeth requiring flap cover

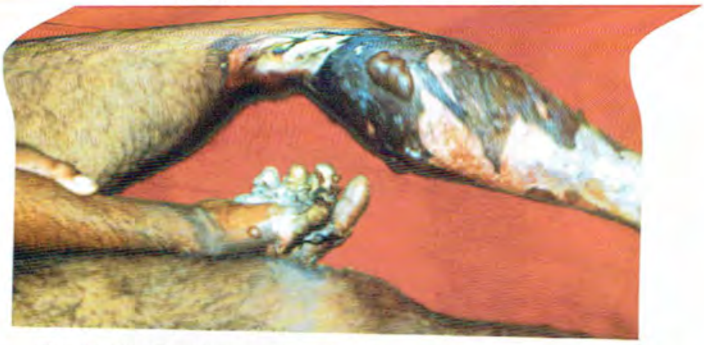


Fig. 21.14: Electrical contact burn on the right hand with full thickness burns on the left leg from clothes catching fire

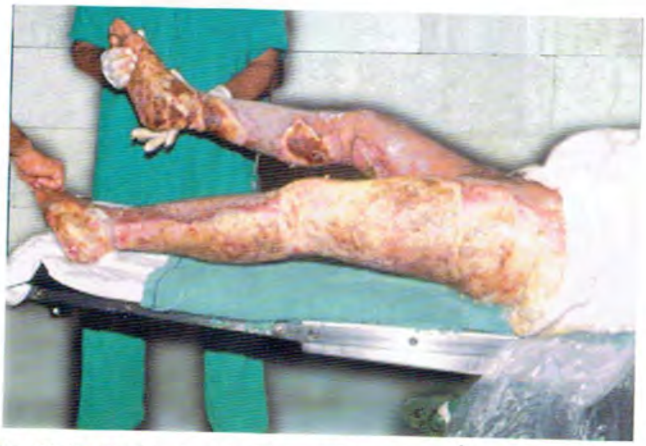


Fig. 21.15: Electrical contact burns of right lower limb showing patchy areas of normal skin with proximal and distal full thickness burns and flame burns of the lower extremity from clothes catching fire

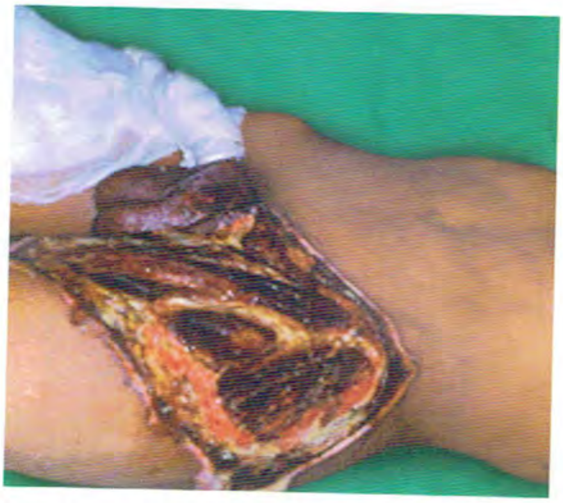


Fig. 21.16: High tension electrical burn involving left groin. The patient was on a pole holding the pole by both the thighs

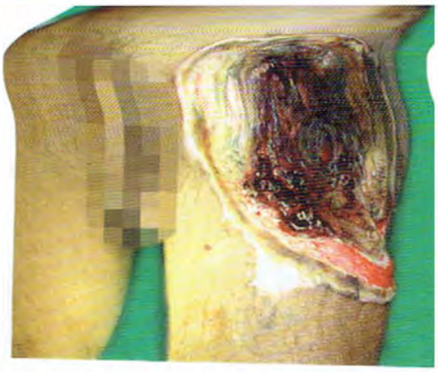


Fig. 21.17: Electrical contact burns on left thigh in ar
sustained while working on an electrical pole with exten
damage exposing hip joint

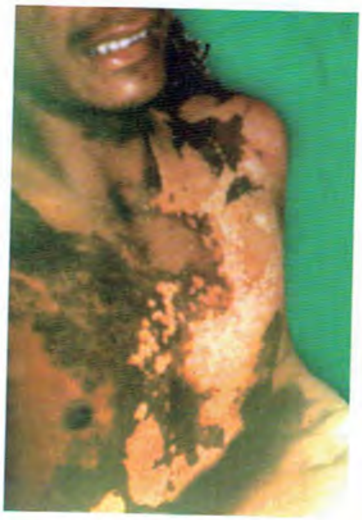


Fig. 21.18: Electrical flash burn

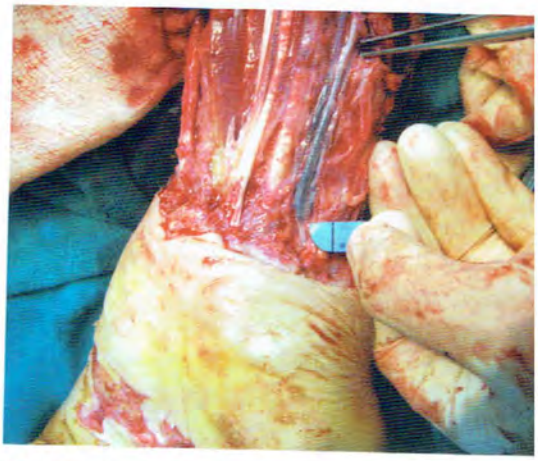


Fig. 21.19: Thrombosed radial vessels following early deb
of electrical burn wound at wrist and lower forearn

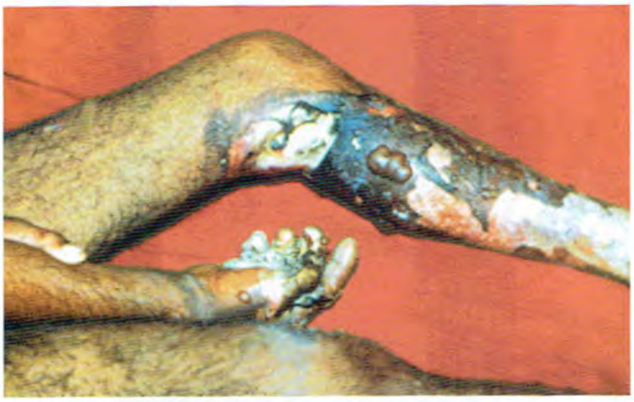


Fig. 21.14: Electrical contact burn on the right hand with full thickness burns on the left leg from clothes catching fire

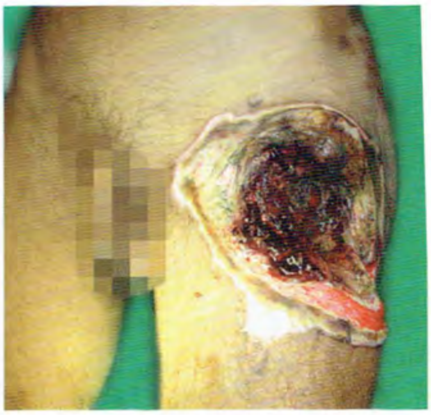


Fig. 21.17: Electrical contact burns on left thigh in an sustained while working on an electrical pole with exten damage exposing hip joint

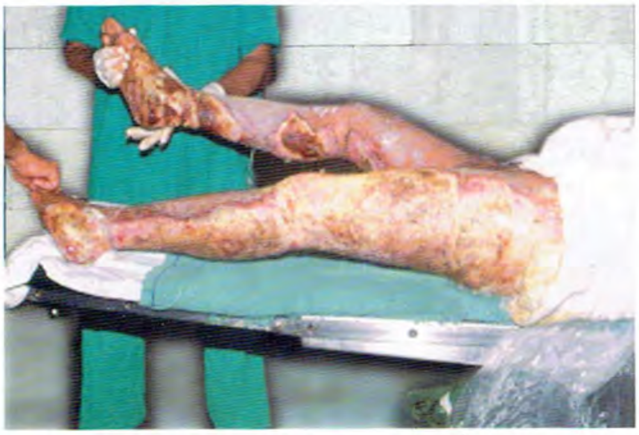


Fig. 21.15: Electrical contact burns of right lower limb showing patchy areas of normal skin with proximal and distal full thickness burns and flame burns of the lower extremity from clothes catching fire

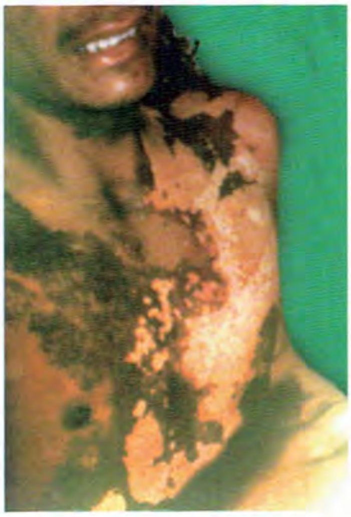


Fig. 21.18: Electrical flash burn

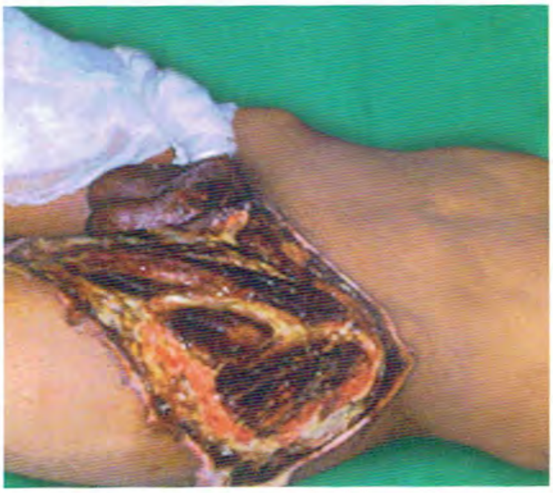


Fig. 21.16: High tension electrical burn involving left groin. The patient was on a pole holding the pole by both the thighs

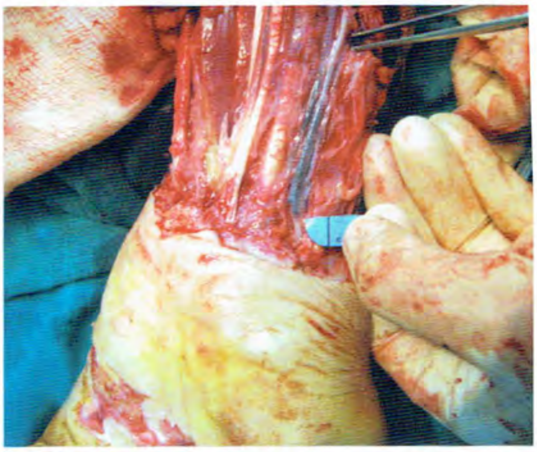


Fig. 21.19: Thrombosed radial vessels following early deb of electrical burn wound at wrist and lower forear

High Voltage Contact Burn[19]

Practically all high voltage contact burns are contact in parallel (as the current passes out from the human body to the ground). There is reduction in the cumulative resistance (resistance of the individual and that of current source). Thus, more current can flow through the human body to the ground leading to more damage to the body tissues. Extent of damage, as previously stated, is related to the duration of current flow, amount of resistance offered to flow, voltage or magnitude of force of current or in summary, the amount of current flown and the rate at which it flows in the human body.

Resistance offered by the skin generates enormous amount of heat leading to evaporation of water content of the skin and underneath tissue. Current flow rises after the initial resistance and peaks up within seconds with complete drying of skin. Current ceases to flow because of carbonization of the skin. This cessation of current is heralded with a flash or arc. Thus, entry point of electric current (high-voltage) appears charred or whitish yellow area with a leathery texture, with markedly depressed central zone surrounded by hyperemic border (Fig. 21.20A). Exit wounds, which can be in lower limb or opposite hand can be multiple and appear ruptured tanned black because of explosive release of heat due to high-voltage current coming out (Fig. 21.20B). Thus, two places show maximum resistance; hence, maximum local damage is seen at these two sites. Once the current crosses the initial resistance and enters the body, current flows through the structure which provides least resistance to current flow. Thus, more current flows through the muscles. This excessive load on the muscle cells which they cannot handle leads to breakdown of muscle cells. This damage is further increased by venous obstruction caused by venous thrombosis and ultimately muscle death from damage to the arterial blood supply. These damages are usually seen in one group or one compartment of muscles. Sometimes these damages are segmental. These segmental muscle damages are seen when the current suddenly moves from one muscle group to another muscle group or where the muscles have multiple points of origin.

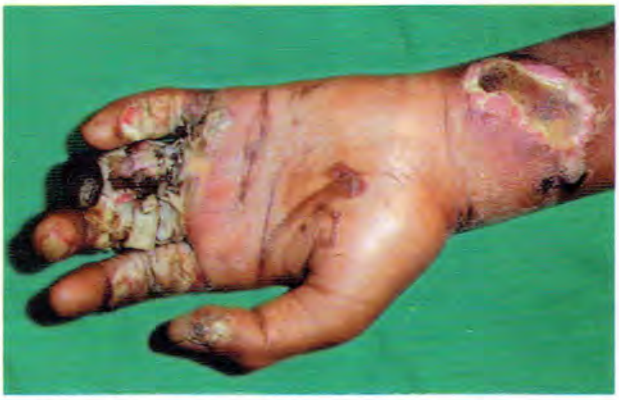


Fig. 21.20A: Entry wounds in fingers due to contact with low voltage current and arc injury at wrist causing full thickness burn at wrist

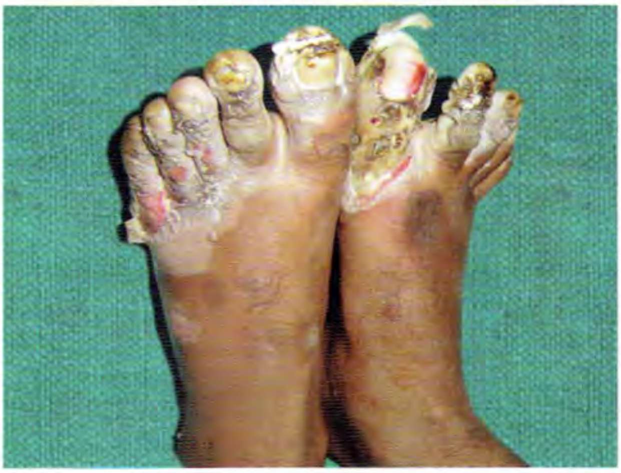


Fig. 21.20B: Exit wound in feet

It is not common to see deep muscle damage much beyond the point of entry with no superficial muscle damage. In the case of upper limbs where entry point of current is the finger, skin damage is seen at wrist, elbow and at the axilla. This type of patchy skin damage is often described as "arcing of current" from finger to wrist and from wrist to elbow because of muscle contraction (Fig. 21.21). It means these damages are indirect damage where current has flown from outside with air pocket in between, i.e. 'jumping of current' across a flexed joint. As the current exits and re-enters the skin, a tremendous amount of heat is generated and the cutaneous injury in flexor creases can vary from minor erythema to explosive charring. However, in the author's opinion, after examining more than sixty cases of high tension burns, this injury is because of flow of current through the blood vessels and at these points i.e. at the wrist, elbow and axilla, the blood vessels become superficial and these vessels give perforating vessels to the skin. Current transmitted through the blood vessels reach the superficial tissues in the skin and again resistance of skin leads to the heat production and full thickness skin damage occurs. Here, damage to the skin is based on changing direction and flow of current.

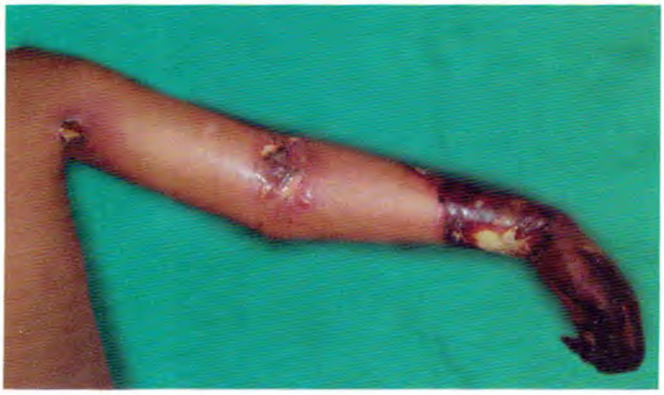


Fig. 21.21: Arcing of current or jumping of current from wrist to elbow to axilla with contact in the fingers

Further damage to the bones and cuff of periosteum has been described. But in a study done by the author on 30 lower extremity high tension burns and 40 upper extremity burns, no such damage was seen, except initially at the point of entry or exit where more damage was seen. From this work, it appears that current flows in a linear axis through the most conductive areas. Nerve damage is seen at the point of entry of current. More often, it is charred and divided. No further nerve damage is seen. However, distant damage is seen in muscles, arteries and veins. More current flows through the muscle because of larger muscle mass and diameter with more water content. Blood vessels because of smaller diameter have more resistance. In arteries, because of rapid flow of blood, there is reduction in current density at any given point while in the veins because of slow flow of blood the current density is high. Therefore, chances of thrombosis of veins are higher which lead to increase in edema and rise in compartmental pressure leading to further damage to the muscles and compression of the arterial supply. The latter may also ultimately get thrombosed leading to complete death of the muscle. It appears that most of the damage in electrical burn is secondary to vascular damage[20] (Figs 21.22 to 21.24).

Electric current can cause tetanic contractions of skeletal muscles or rupture of tendons and at times dislocation and multiple fractures. The heat generated from the passage of current through the bone can cause aseptic necrosis, burns to periosteum or complete destruction of bone matrix leading to autoamputation (Fig. 21.25). The rate of auto-amputation is reported to be as high as 35=60%.[21]

Sometimes, in alternate current with inability to "let go" effect, a limb or a person as a whole may be completely charred from the electrical current and one may not be in a

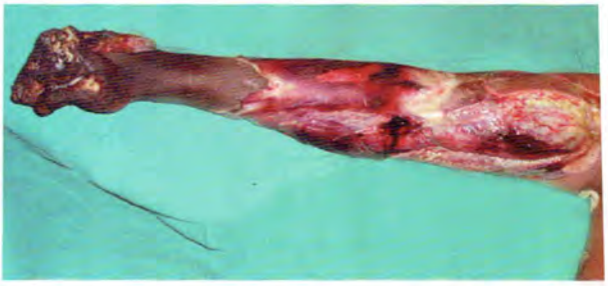


Fig. 21.22: High tension injury showing extent of damage to d
structures beyond the apparently damaged skin.Though the li
demarcation is till elbow, the muscles are seen necrosed much
proximally, thus requiring a higher level of amputation

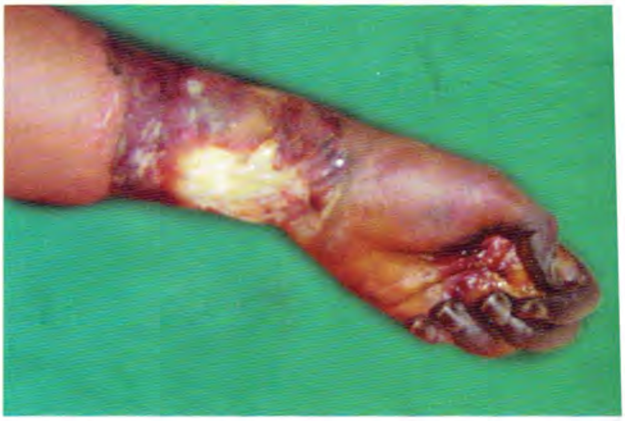


Fig. 21.23: high voltage contact burn in palmar surface of hand
arcing causing full thickness circumferential burns in wrist and lo
forearm causing compartment syndrome leading to ischemic dam
to all fingers

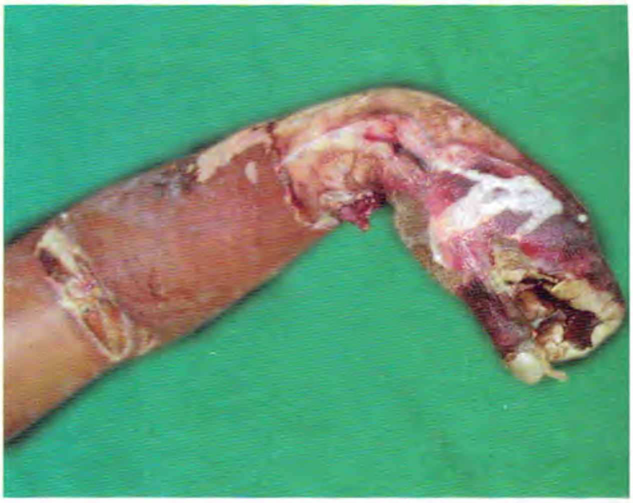


Fig. 21.24: High voltage current causing primary gangrene of ha
with arcing at elbow causing full thickness burns over elbow

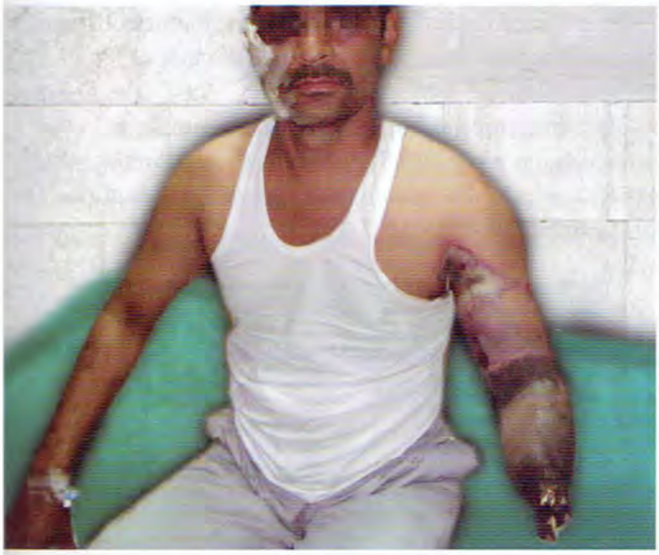


Fig. 21.25: Primary forearm autoamputation following high voltage injury with visible proximal damage of elbow extending till axilla

condition to identify anything separately. Some peculiar injuries are seen in electrical burns from the point of entry of electrical current. These are hollow-viscous or solid viscous injuries. Usually intra-abdominal injury is prevented by a comparatively avascular peritoneum. However, when current enters through the abdomen one gets direct injury to the intestine and colonic injuries are the most common (Fig. 21.26). Similarly, injuries to the urinary bladder and penis also take place from direct current damage.[22] Fatal injuries to the liver in a similar fashion have been described[23] (Fig. 21.27).

Thus, anatomical positions have great bearing as direct damage to any organ can be affected by the electrical injuries. It is not necessary that structures are involved between entry and exit points as path taken by the current may markedly vary and many structures may completely survive as current may jump from one group of anatomical structure to the other which is in close proximity.

SEQUELAE OF SECONDARY EFFECTS AFTER INJURIES

Because of an electrical injury there is marked extravasation of fluid from the muscles leading to rise in intra-compartmental pressure leading to further decrease in the venous outflow, which impairs arterial inflow and causes further devascularization of muscles and other tissues. If this compartment pressure is not released quickly, damage to the muscles and other structures becomes irreversible leading to gangrene of the limbs.

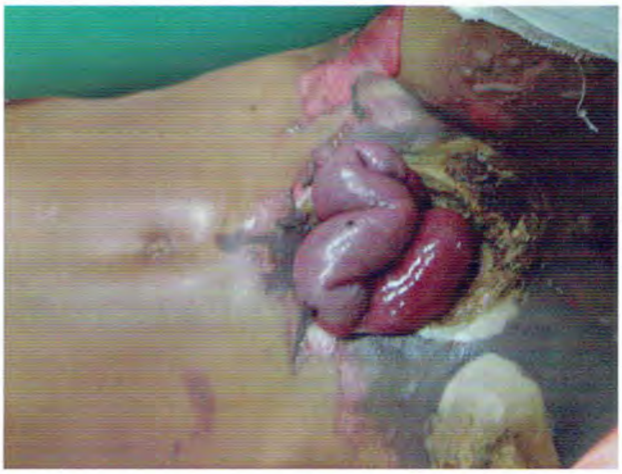


Fig. 21.26: A 14-year-old boy with electric contact burn over lower abdomen exposing the bowel with loss of external genitalia

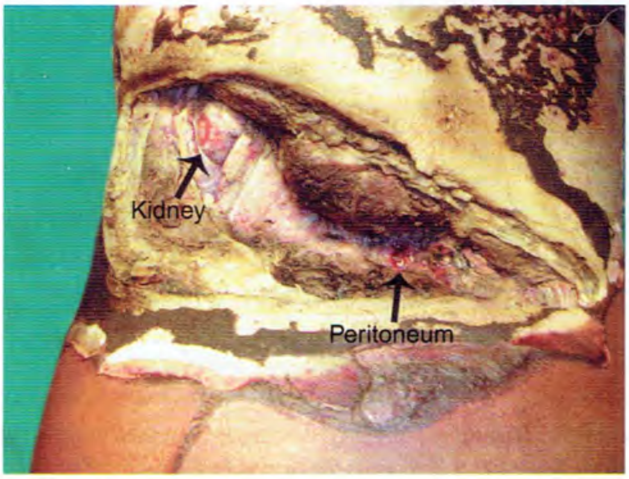


Fig. 21.27: Deep high voltage injury in trunk with extensive damage of all abdominal muscles exposing underlying viscera

Damaged muscles release pigment so do the damaged red cells. These pigments enter into the blood and can block the renal tubular system leading to acute renal failure. Another mechanism, which is not well understood, is the release of substances by ischemic muscles which enter into the blood. These substances have a depressive effect on the myocardium. This is well seen by improvement in cardiac functions once an injured muscle or limb is removed by amputation.

DIAGNOSTIC MODALITIES[24,25]

Though many diagnostic modalities have been suggested to diagnose the muscle damage which includes various enzyme levels, SGOT, SGPT but all these are non-specific as these may be elevated without muscle damage also. Presence of pigment in the urine is the single most important indicator of muscle damage.

The patient should be placed on a cardiac monitor and a 12 lead ECG should be obtained. MRI can be used to detect edema but is not necessarily of much help.[26]

A Technetium Tc 99m Stannus Pyrophosphate (99m Tc Pyp)[27] muscle scan can be safe, sensitive, noninvasive and reliable diagnostic tool in defining the viability of muscles. Areas of muscle unable to show uptake of isotopes (cold spot) are indicative of dead muscle. However, the test is extremely sensitive and many false positive effects may be seen when there is increase in compartmental pressure. In case of severely mummified tissue requiring amputation, the exact level of vascular occlusion can be localized by preoperative arteriography and the approximate level of surgical amputation can be decided.[28]

Exploration, direct visualization of muscle, its contractility and bleeding on cutting are the practical tools in deciding the level of muscle damage.

TREATMENT

In the emergency department, attention to the protocol of basic life support and advanced life support should be followed as per requirement. Tetanus prophylaxis is essential for all electrical burn patients. Hydration should be started with ringer lactate. Surface damage in electrical injury may not necessarily reflect the severity of deeper tissue damage so fluid replacement may be grossly underestimated. Resuscitation of electrical burn patient from the point of view of hypovolemia using conventional skin burns formula may not be correct as most of the fluid is lost in the muscles. Here, fluid should be titrated so that one can get a urine output of 2 ml/kg body weight/hour. The fluid required in an electrical burn may be 2-3 times more as compared to thermal burns. The objective of aggressive early rehydration is to avoid renal failure secondary to myoglobinuria as there is release of myoglobins from damaged muscle cells and small amount of hemoglobin from damaged red blood cells and unidentified intracellular substances which can produce severe metabolic acidosis.

In the presence of pigments in the urine, alkalinization of urine should be done and mannitol should be give so that tubules can clear the pigments. Initial dose of 25 gm of mannitol followed by 12.5 gm/hour for several hours usually produces urine volume of 200-300 ml/hour. This further forces more fluid to be infused in electrical burns. The persistence of reddish black urine once the urine flow 50 ml/hour indicates massive muscle damage (Figs 21. and B).

In these cases, the arterial pH may remain below after volume repletion. Slow intravenous administratio 80-120 ml of 8.4% soda bicarbonate/hour is continue pH is stabilized to above 7.35.

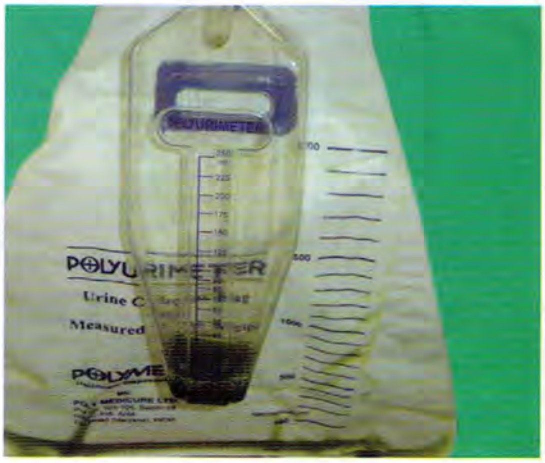


Fig. 21.28A: Myoglobinuria indicated by reddish black urine req vigorous resuscitation

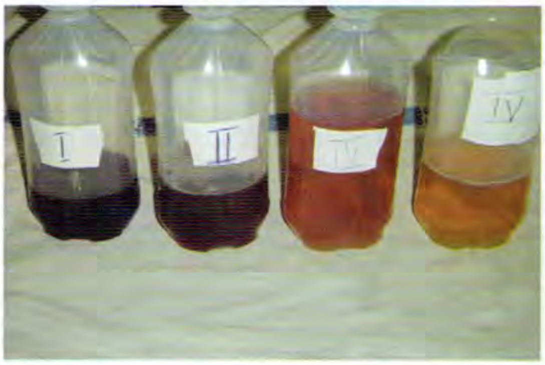


Fig. 21.28B: Gradual improvement in both color and quantity of with proper resuscitation, osmotic diuresis and alkalanization of

Local Wound Management

In electrical flash burns, where there are only sur thermal burns, the standard treatment of clean debridement and topical dressings using antimicrobial ag is sufficient. When deeper structures are involved, t may be significant limb edema predisposing the patie the danger of compartment syndrome. In these patient is necessary to perform a fasciotomy as early as possib salvage the limb (Figs 21.29A to 21.30B). This is because, if the underlying necrotic muscle masses go undetected, there are chances of gangrene of distal extremities as well as early toxicity (within 3-5 days) and later infected abscess formation producing lethal sepsis. Likewise in circumferential full thickness limb burns, an escharotomy may be necessary to eliminate the constricting effect of the circumferential injury. It is important to resuscitate the patient well prior to fasciotomy. Once the fasciotomy is done, the tourniquet effect of deep fascia due to underlying edematous tissue is released and the venous return is established from the distal tissues, the myoglobin pigment from these ischemic distal tissues enters into the circulation. The pigments then go on to block the renal tubules and the patient may end up in renal failure.

Surgical exploration is the only reliable method of identifying the presence of muscle damage. Early excision or debridement should be done to prevent further damage and to reduce the immediate depressive effect of dead tissues on the myocardium. Many workers have suggested a policy of 'wait and watch' for development of line of demarcation. This policy may lead to an increase in the secondary damage while early surgical interventions have no side effect other than the one that the patient may require a second excisional surgery.

Amputation: The large necrotic muscle mass and dead tissue resulting from the passage of electrical current should be removed early. The initial fasciotomy often limits further ischemic damage to distal tissues by removing the effects of vascular compression and thus lowers the site of amputation and ensures least number of complications and better survival of body parts and life as well. In general, necessary amputations can be done safely after 48-72 hours of injury once the patient has been thoroughly resuscitated and line of demarcation is established. These early amputations should be preferably done under regional blocks as the myocardium remains sensitized to various drugs for 5-7 days following passage of electrical current through the heart (Fig. 21.31). General anesthesia should be avoided. At times the level of amputation may be higher than the cutaneous line of demarcation because the underlying muscle damage extends more proximal than the skin damage. Infact for several inches proximal to the line of demarcation, the skin may appear normal and yet the underlying muscles may be dead. Hence, they may require revision more than once to a higher level.

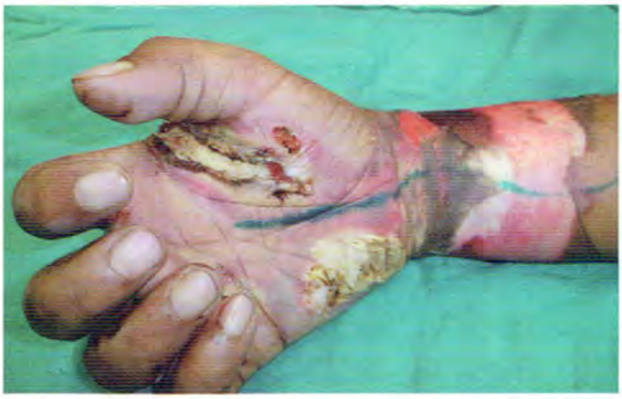


Fig. 21.29A: Contact injury to hand and wrist causing proximal compartment syndrome in forearm and tight carpal tunnel

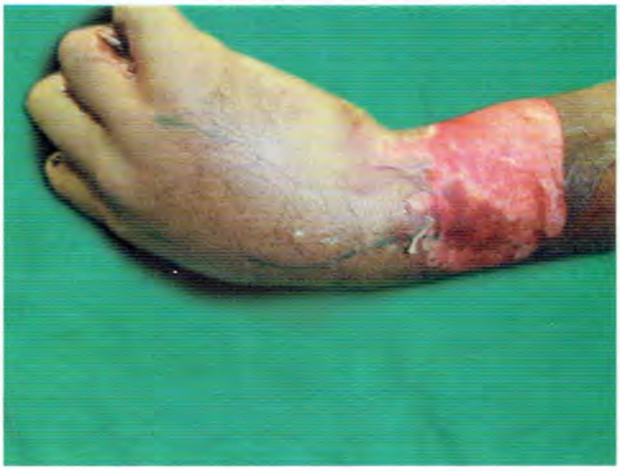


Fig. 21.29B: Dorsum of same hand showing circumferential injury at wrist with edematous dorsum

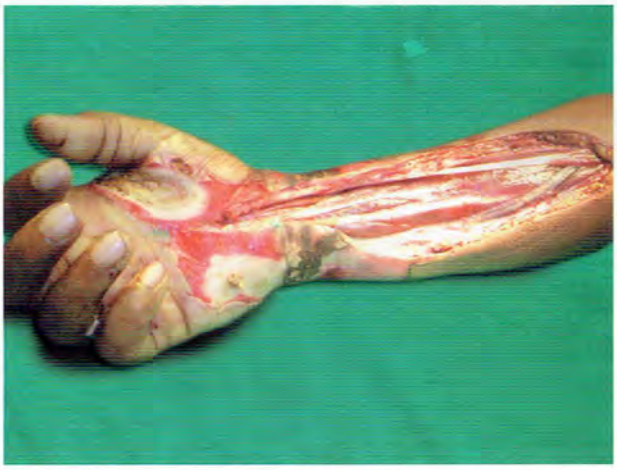


Fig. 21.29C: Early fasciotomy done to release compartment syndrome and opened carpal tunnel showing thrombosed ulnar vessels

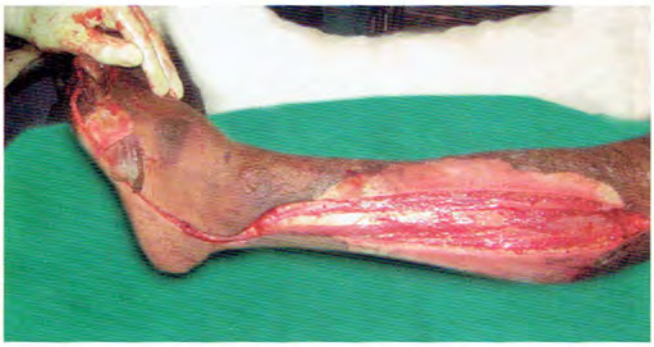


Fig. 21.30A: Early fasciotomy in lower limb to release compartment syndrome

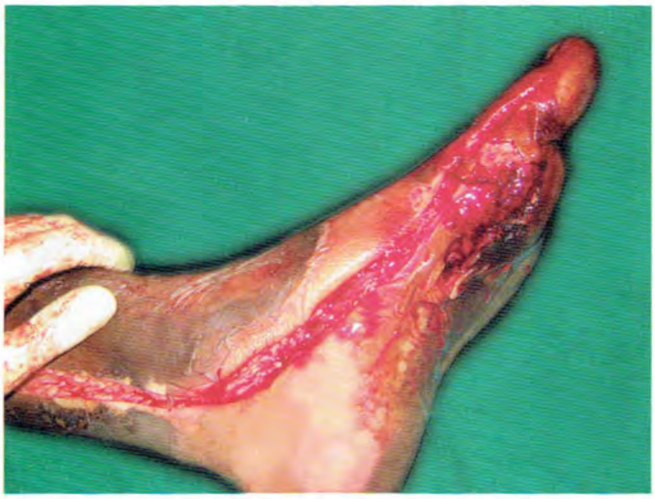


Fig. 21.30B: Fasciotomy incision extending into foot

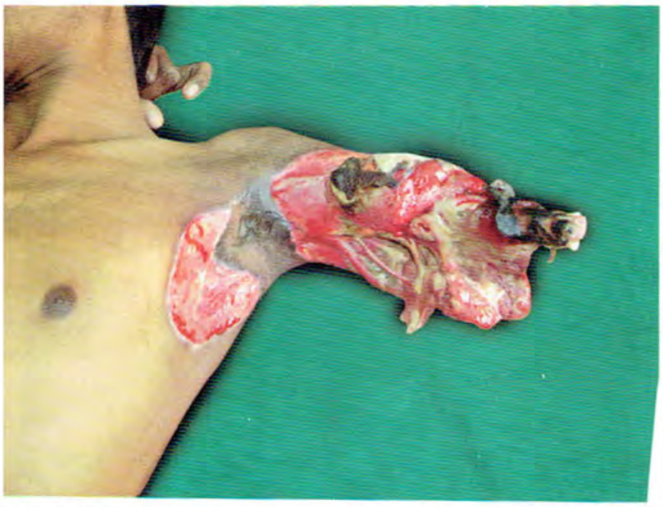


Fig. 21.31: Guillotine amputation done in midarm level showing residual dead muscles requiring revision of amputation

Ligation of subclavian vessels may be essential in case the entire length of upper limb is gangrenous and requires amputation at shoulder level so as to avoid blow out from axillary vessels at amputated site if the damage extends till axilla or more proximal (Figs 21.32A to 21.34).

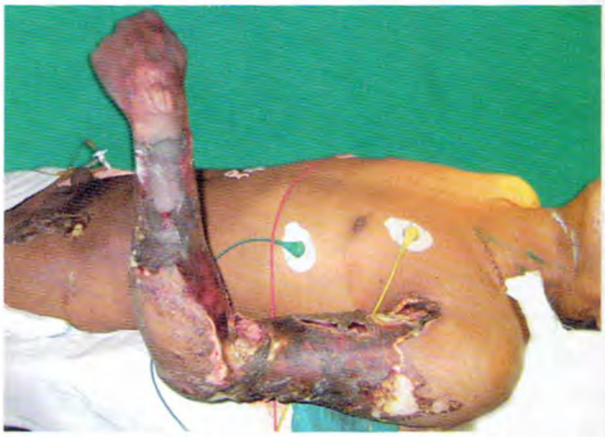


Fig. 21.32A: Gangrene of entire length of upper limb because of hig voltage contact burns requiring amputation at the level of shoulders Prior to amputation the subclavian vessel ligation was required so a to prevent their blowout postoperatively

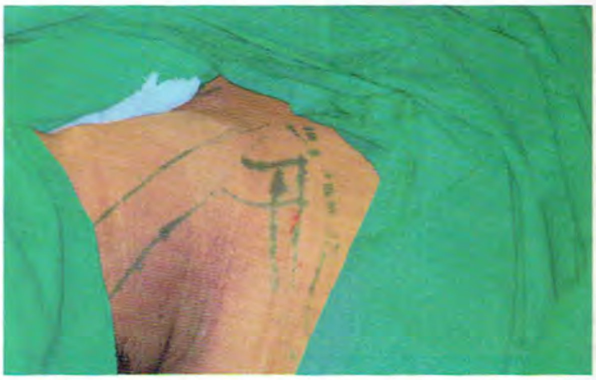


Fig. 21.32B: Marking for locating subclavian vessels

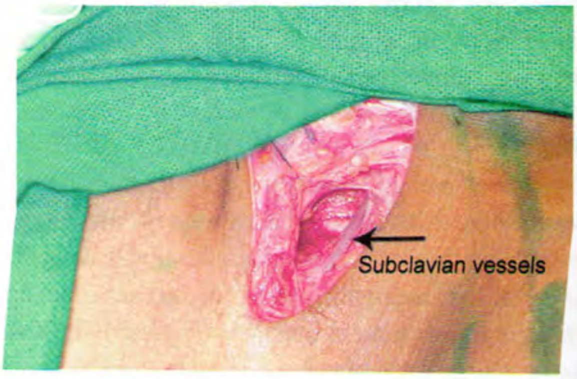


Fig. 21.32C: Subclavian vessels exposed beneath the clavicle

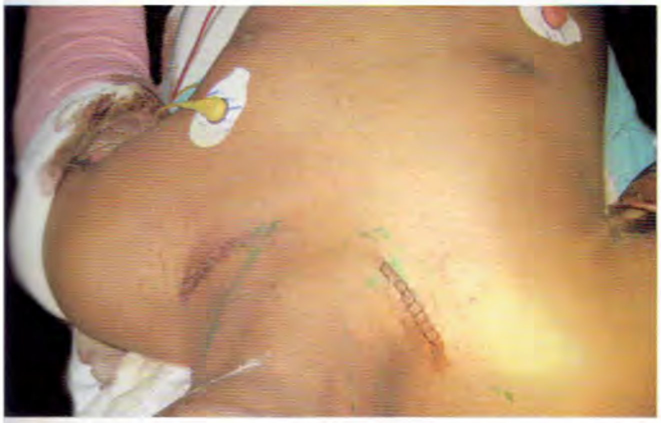


Fig. 21.32D: Bilateral subclavian ligation achieved after which both the gangrenous limbs can be disarticulated safely at the shoulder

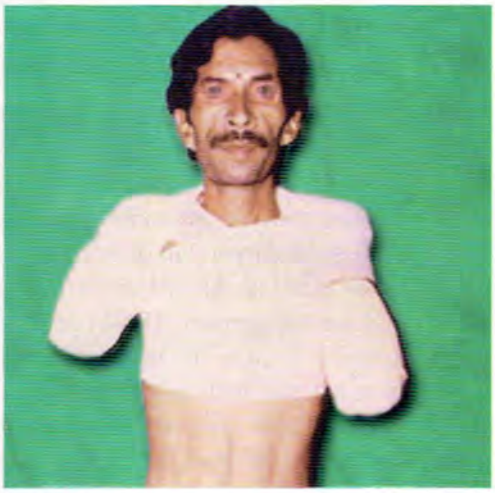


Fig. 21.33: A case of arm to arm transmission of high voltage electricity leading to above elbow amputation of both the upper limbs

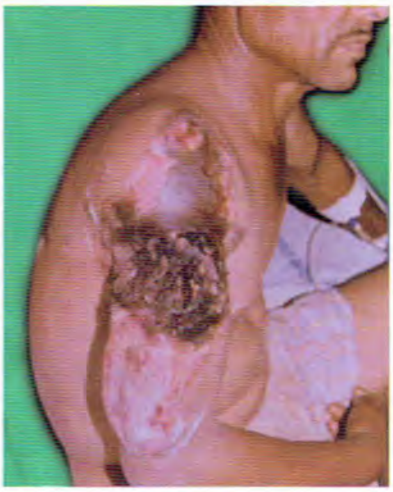


Fig. 21.34: Electric contact burns in series, no wound of exit or grounding. Maximum local damage by production of heat

COMPLICATION OF ELECTRIC BURNS

Some specific complications/effects of electrical burns[43] are as follows:

Neurological Complications[29-32]

Many neurological complications have been described in electrical burn. Some are understandable while others are not fully understood. Electrical current can cause acute and delayed injury to the central and peripheral nervous systems. Acute central nervous system symptoms include altered mental status like confusion, impaired concentration, and disorientation. More severe cases may have paralysis, seizures, coma, loss of consciousness and depression of respiratory center. Injury or effects on peripheral nerve, median, ulnar or radial and sometimes compound global brachial plexus injuries can also take place because of severe muscle contraction pulling the brachial plexus. Mostly, injuries to the nerves are confined to the local site where a nerve is directly damaged from the electrical current. In some cases the nerve damage is secondary to increased intra-compartmental pressure. The effects revert to normal once the pressure is released. When irreversible damage occurs, nerve grafting may be required. However, results of nerve grafting may not be good because of intense scarring in the region of damage and also because of the associated muscle damage.

Damage to brain and spinal cord has also been described[39-43] (Fig. 21.35). The cause of this damage is not well understood. Often, a hemorrhagic infarct is seen in brain especially in the area of thalamus and internal capsule. These are the areas where blood vessels are less protected in the white matter and are more likely to have flow of current outside leading to damage. These cases develop extra-pyramidal symptoms.[31,32] Probably similar effect is seen in the spinal cord leading to paraplegia or quadriplegia. Delayed effects in the spinal cord have also been described which develop months and years after an electrical insult. In these patients, motor symptoms are more pronounced than the sensory involvement. Mechanism of its development is not fully understood but appears to be due to slow development of cord ischemia.

Cardiovascular and Pulmonary Complications

Both heart and lungs are well protected by comparatively avascular structures, i.e. pericardium and pleura.

Different conduction abnormalities are seen in the heart. Cardiac arrest can be either complete standstill (asystole)

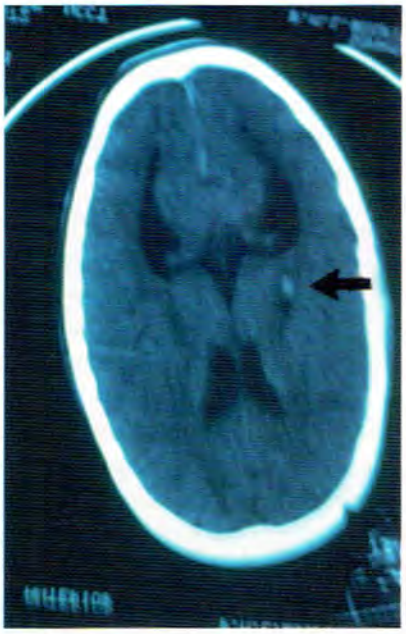


Fig. 21.35: CT Scan showing hemorrhagic infarct in the area of internal capsule following electric contact burns

or ineffective overactivity (ventricular fibrillation). Different levels of heart block have also been described. Most common types of conductive anomalies are sinus tachycardia, ST elevation and T-wave depression. Most of these revert to normal in due course of time. Multiple extrasystoles and atrial fibrillation can also develop requiring administration of different cardiotropic drugs. Most of the cardiac effects recover spontaneously, long-term consequences being very rare.

Enzymatic assessment of cardiac damage may not be helpful because most of these are elevated due to striated muscle damage and may not be cardio-specific. LDH level changes may also be seen without ischemic changes in the heart as slight damage of cardiac muscle recovers spontaneously and may not be of any consequence.

Delayed vascular damage has also been described.[33] Whether one should describe them as true electrical cause or secondary hemorrhage is a point of individual perception. However, one must remember that small vessels are likely to be affected because they provide more resistance to the flow, produce more heat and develop thrombosis. If there is extensive necrosis over large blood vessels, there is always a danger of delayed hemorrhage from these large vessels. This can be minimized by providing early vascularized muscle flap cover over these areas which gives additional blood supply to the vessel wall.

Lung tissues are usually not involved. The most s pulmonary sequel from an electrical injury is respi arrest. Usually, it is the pleura which gets damaged l to effusion or collapse of lung necessitating inter drainage.[34] Rarely atelectasis, pneumonia, hemopn thorax, pulmonary contusion and laryngeal injuries n seen.

Eye

Different eye changes have been described which i damage to cornea, retina or optic nerve. The most co involvement is of the lens leading to early developm cataract.[35] It may be on one side or both eyes m involved. It usually manifests in 6 months to 1 yea electrical injury to head. An ophthalmologist's interv is required. One must remember that phacoemulsifi may not be an easy procedure in these cases (Figs and 21.37).

Bone and Joint

Bone and joint injuries may be associated with ele burns. These may be secondary to fall or because of and strong muscle contraction. An orthopedic consu is required for optimum management. However, all th tissue must be excised if fracture site is in the area of and/or areas must be covered with vascularized flap (regional flap or a free flap). Free flap must be car planned and anastomotic site should be placed awa the site of injury.

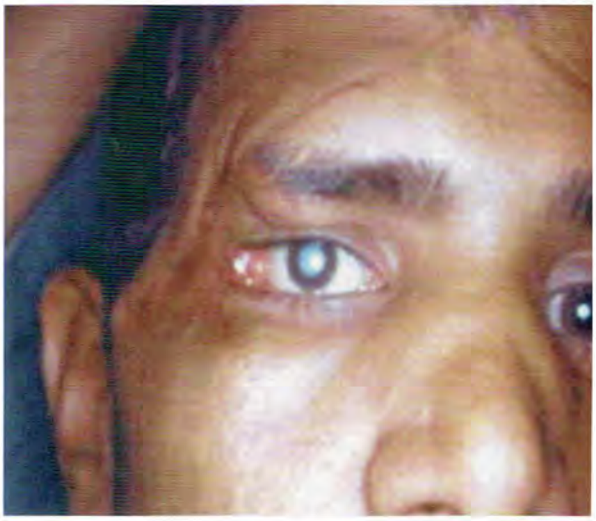


Fig. 21.36: Cataract development after electrical contac burn near zygoma

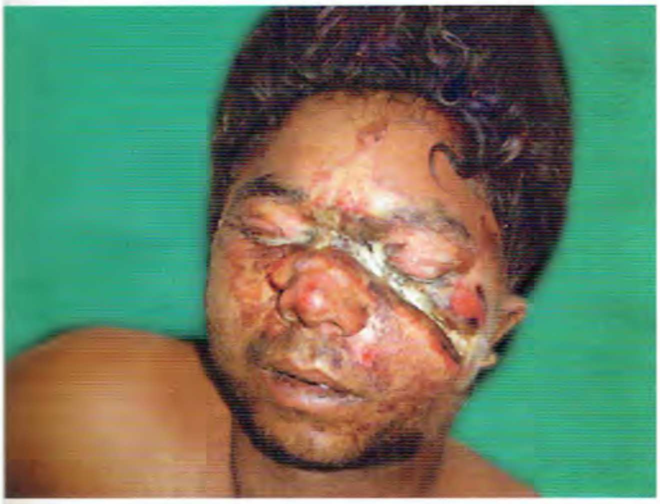


Fig. 21.37A: Contact injury on face by wire falling accidentally causing deep injury on nose, left eye and cheek with total loss of left eye

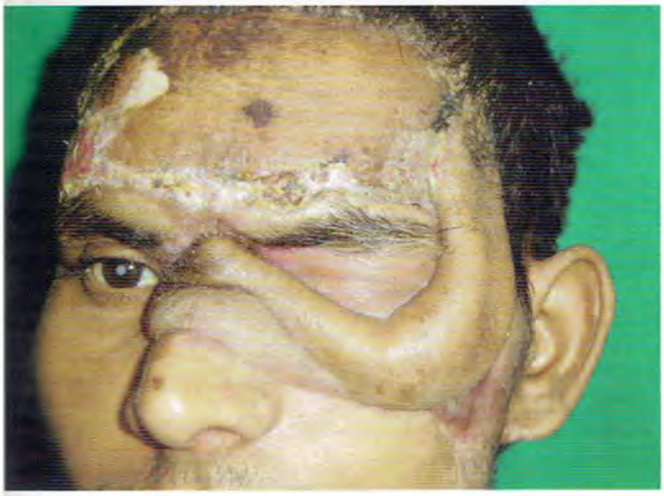


Fig. 21.37B: Following debridement laterally based forehead flap cover given to cover the defect and cataract seen to have developed in the contralateral eye

Gas Gangrene and Tetanus

Apart from damage and destruction of tissues of the body, infection of the wound may occur leading to sepsis especially if the gangrenous limb or tissue is not removed timely. Gas gangrene and tetanus due to clostridial infection in the dead tissue has been reported following electric burns. Gas gangrene require urgent management, i.e. amputation of the involved limb in order to save the patient's life along with medical management in the form of appropriate antibiotics (Penicillin) and anti-gas gangrene serum.

BURNS OF SPECIAL SITES

Scalp (Figs 21.38A to 21.39B)

Scalp is third most common site involved in electrical burns after upper and lower limbs. All scalp burns are full thickness. Invariably, there is involvement of calvarium as well. However, involvement of dura is the least common. These burns may have different degrees of brain involvement. Most commonly when brain is involved there is a localized hemorrhagic infarct especially in the white matter areas like internal capsule or thalamus as vessels of these sites are comparatively unprotected.

Management

When localized to scalp with no other injuries like cardiac, renal and pulmonary involvement and the patient is otherwise stable, they are treated by primary excision of burned scalp and covering the defect by transposition flap or rotation flap or by the free flap (latissimus dorsi flap with skin graft). These vascularized flaps provide vascularity to the bone underneath and may allow it to regenerate. Alternatively all obvious dead bone is removed till bleeding areas or both tables are removed. Reconstruction is done with either bone substitute or by split calvarial bone or rib graft covered with a flap.

In more serious patients, who cannot be taken up for surgery early, these areas are permitted to granulate and later skin graft is applied over the granulating area.

Abdomen (Fig. 21.40)[36,37]

These burns involve major portions of abdominal wall because of the low water content of the peritoneum. Abdominal contents are normally saved by the resistance of the peritoneum. However, in case of direct prolonged contact, local damage to the hollow-viscous may be seen. There can be necrosis of intestines and small bowel perforations or even direct electrical damage to liver, pancreas, gallbladder, etc. which can be fatal. Stress ulcers, Curling's ulcers and gastrointestinal bleeding have also been reported. Treatment consists of excision and exploratory laparotomy in case where there are signs of intra-abdominal injury which is confirmed by ultrasonography and CT scan. Injured hollow-viscous are treated by colostomy or exteriorization of the colon or small bowel. In case there is severe injury in perineal region, it can lead to loss of male external genitalia as well as bladder injury. These have to be reconstructed using local or regional flaps.[38-40] There are

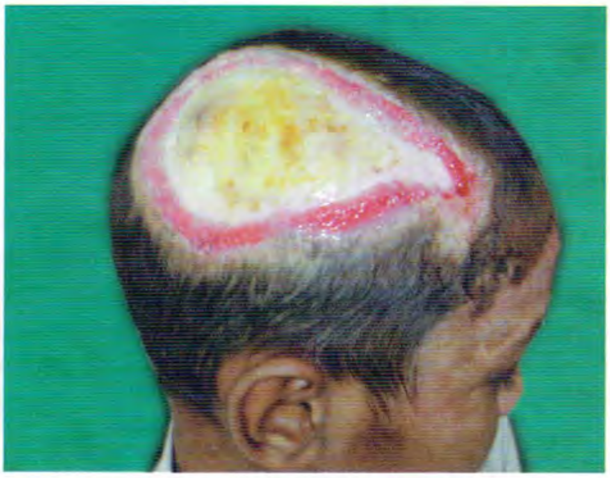


Fig. 21.38A: Dead exposed skull bone due to high voltage injury to skull

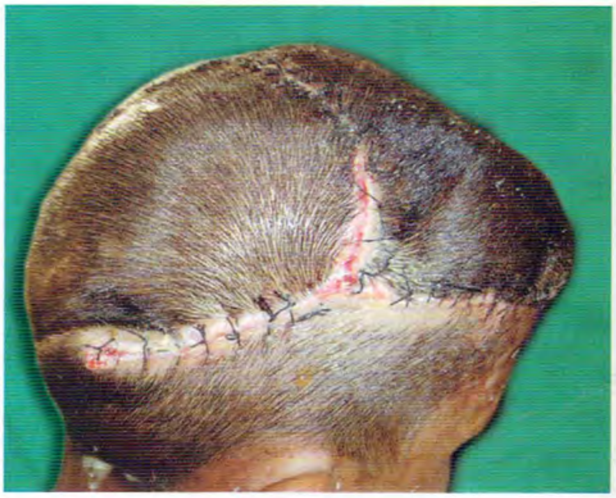


Fig. 21.38D: Adjacent scalp flap transposed to cover the defect

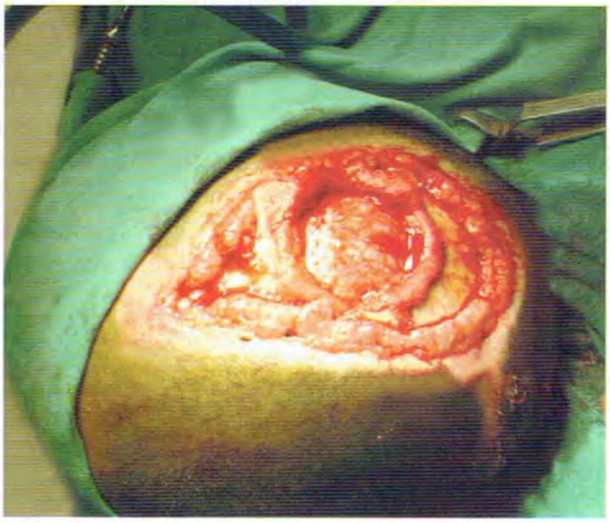


Fig. 21.38B: Dead outer and inner table debrided exposing the dura

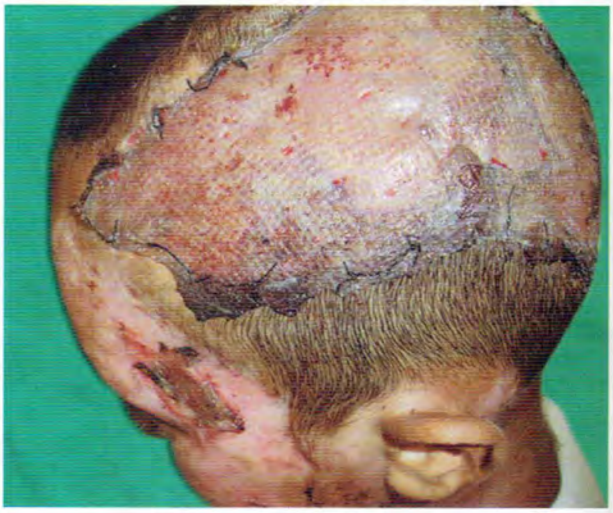


Fig. 21.38E: Donor area grafted

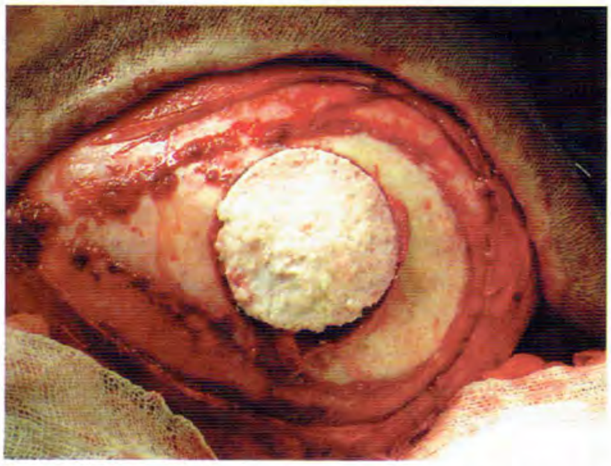


Fig. 21.38C: Bony defect covered with artificial bony material

various reports of successful treatment of such rare injuries and reconstructing abdominal wall defects using different flaps e.g. free latissimus dorsi flap, tensor fascia lata flap etc[41] (Figs 21.41A to D).

Upper Extremity and Lower Extremity (Figs 21.42A to 21.46B)

Involvement of upper extremity is the most common injury of the electrical burns. Linear flow of electrical current may cause muscle damage right up to axilla, however this damage may be to a particular group of muscles only. These group of muscles should be excised primarily and all areas should be covered with a vascularized flap. In the author's opinion this probably prevents further damage to the other group of

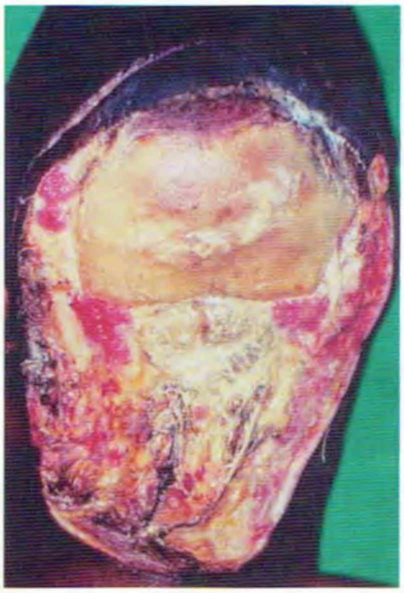


Fig. 21.39A: Post electric burn exposed skull and neck tissues

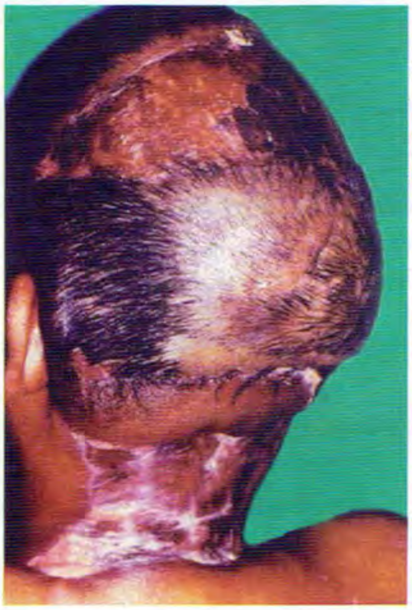


Fig. 21.39B: Transposition flap cover from adjacent scalp

muscles by providing alternating venous channels for venous return. Most commonly used flap is latissimus dorsi muscle flap which is invariably not involved in upper limb electrical injury. However, when all muscles with their neurovascular bundles are involved, a prompt amputation should be preformed. Unfortunately upper limb amputation is 2-3 times more common than the lower limb despite reconstructive procedures to salvage the limb.

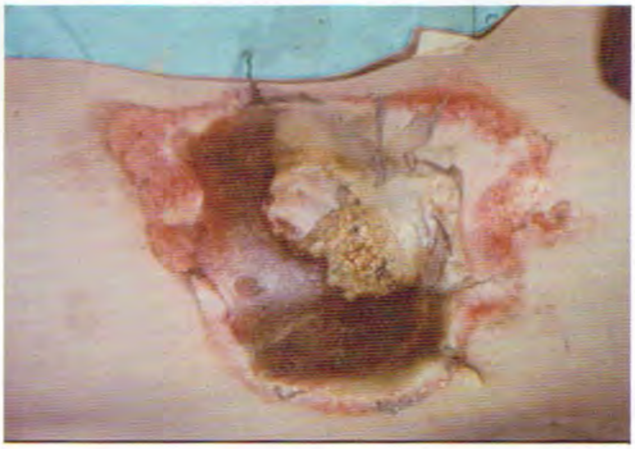


Fig. 21.40: High tension electrical burn of abdomen wall. Patient climbed on the roof top of electrified train

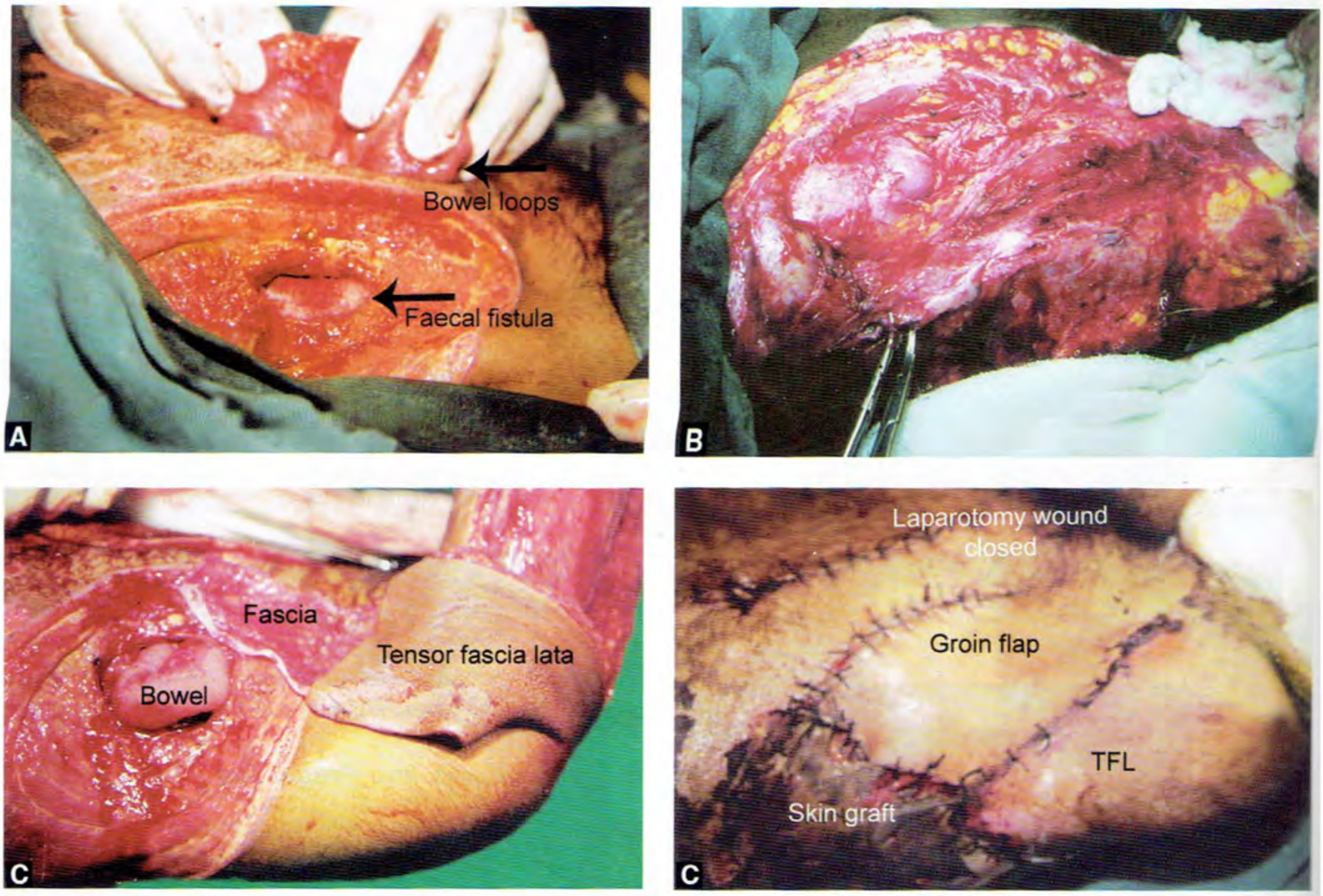
In the case of lower extremity with larger muscle mass, more dead muscles produce a greater effect on the heart. Therefore, one should not hesitate to amputate the lower extremity to save the patient as good lower limb prostheses are available for patient rehabilitation.

Electrical Burns and Pregnancy

95% of electrical burns are in men. Therefore, involvement of pregnant women is very rare. However, whenever it happens, invariably there is death of the fetus. It is not known whether it is because of the direct current on the fetus or due to secondary effect from burns to the mother. It is a well known that nature always gives priority to the mother over the fetus and the latter is expelled out to save the mother. Thus, pregnant women with electrical burn abort or expel their fetuses in natural way.

SUMMARY

This chapter summarizes the current concepts in the management of patients with electrical injuries. Apart from understanding the pathophysiology of manifestations of these burns, both local and systemic, an early diagnosis of the extent of underlying tissue damage is equally important. An aggressive program of prompt and adequate resuscitation, timely fasciotomy, and early debridement including amputations and vascularized flap cover can decrease the incidence of renal failure, early toxemia and septicemia reducing the morbidity and mortality. The importance of progressive destruction of tissues in electric burns cannot be overlooked and necessitates repeated assessment and frequent debridements. Following healing of all wounds,



Figs 21.41A to D: (A) Case of faecal fistula following electrical contact injury on abdomen. Patient taken up for laparotomy —Bowel delivered from laparotomy wound. (B) Resection anastomosis of diseased bowel done. (C) Extended tensor fascia lata flap raised to cover exposed bowel site but was insufficient. (D) Laparotomy wound closed and defect covered by TFL and groin flap

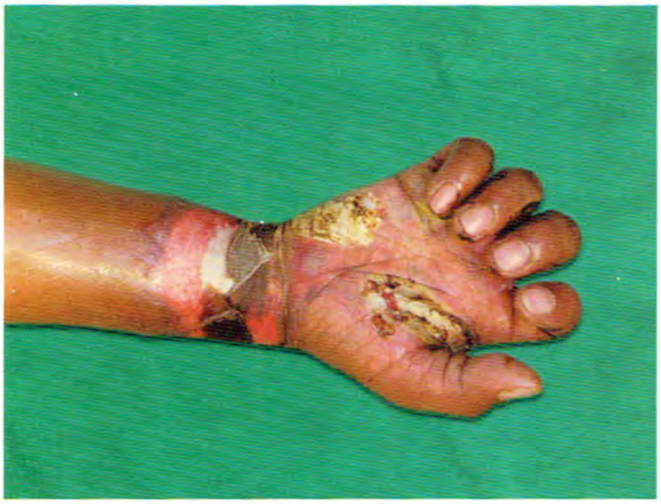


Fig. 21.42A: Full thickness burns over wrist following contact with current in palm

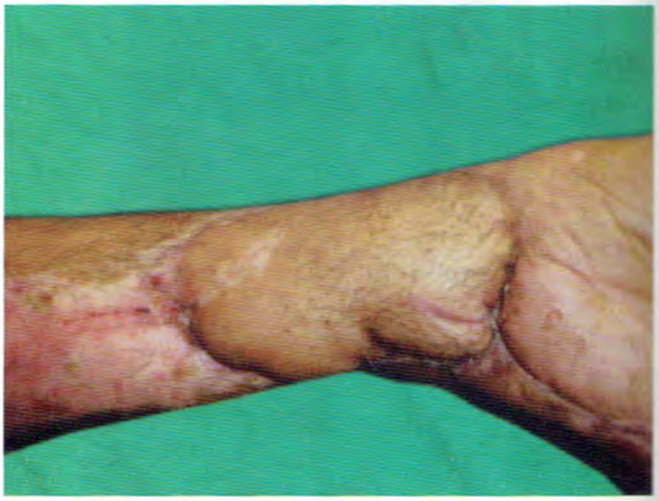
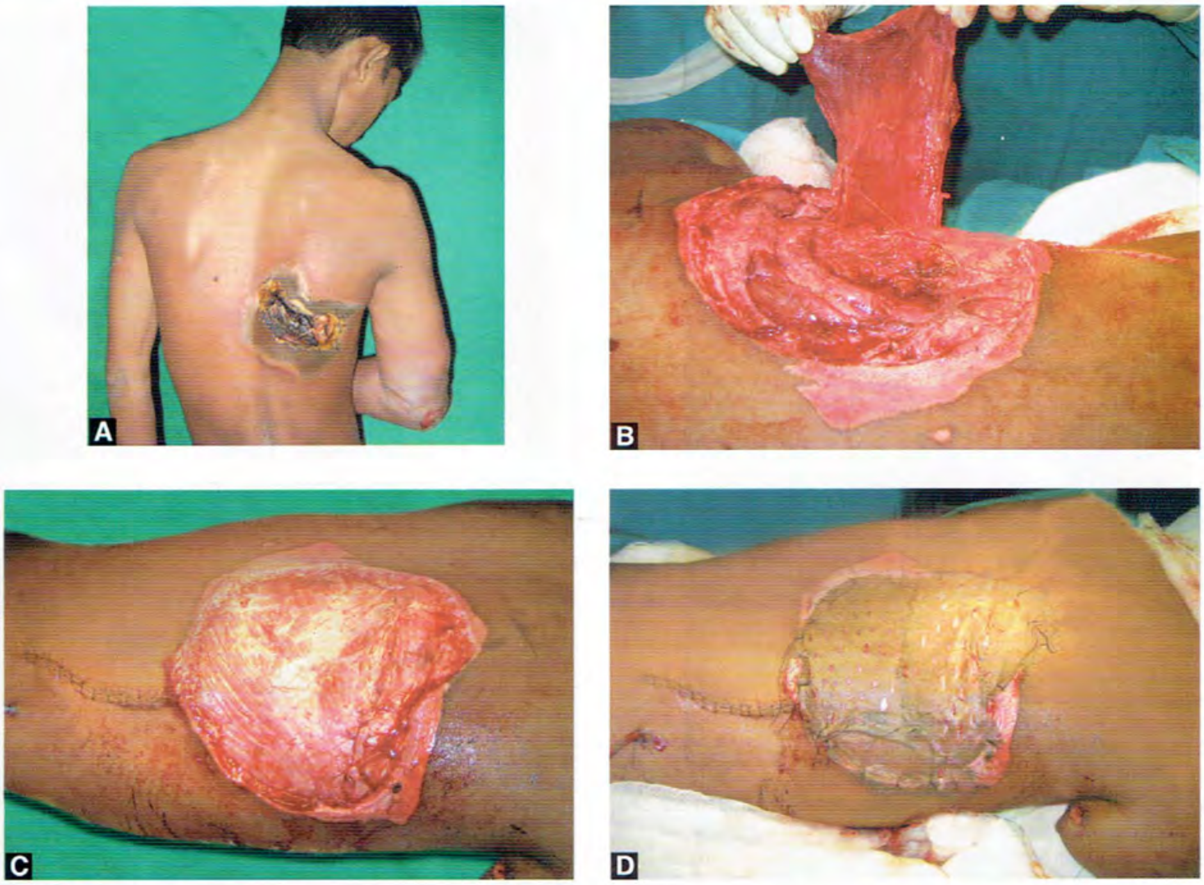


Fig. 21.42B: Wound debrided and exposed tendons and nerves covered by groin flap



Figs 21.43A to D: (A) High voltage injury on the back of chest. (B) Wound debrided and exposed ribs with raised latissimus dorsi flap. (C) Latissimus dorsi flap covering the defect. (D) Skin graft applied over muscle flap

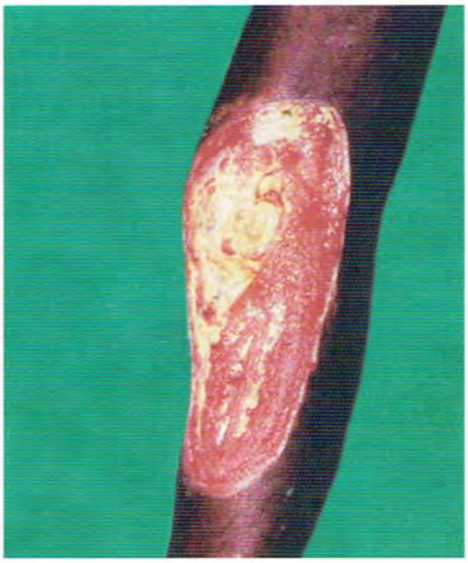


Fig. 21.44A: Exposed knee joint following electric contact burns

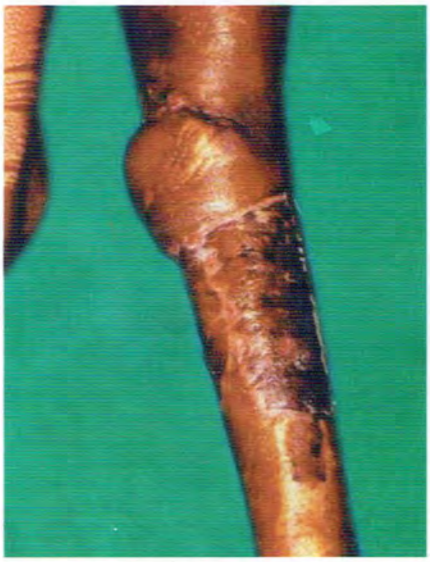


Fig. 21.44B: Covering knee joint by superiorly based fasciocutaneous flap

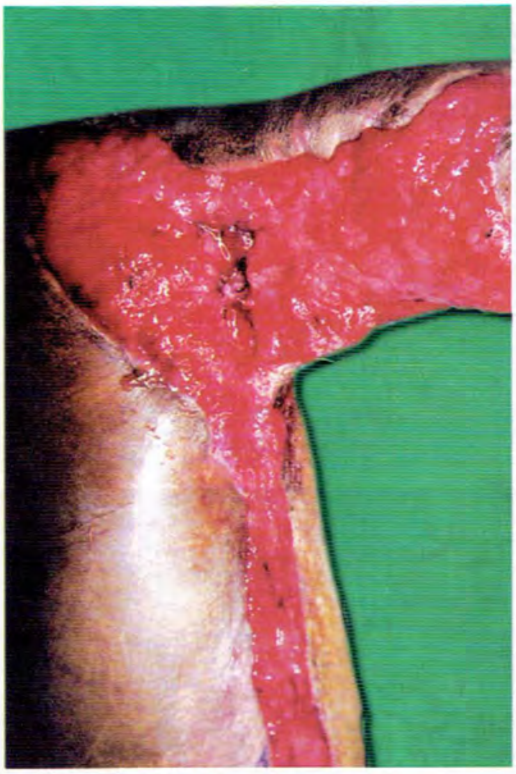


Fig. 21.45A: Exposed axillary vessels following debridement of eschar from electric burns

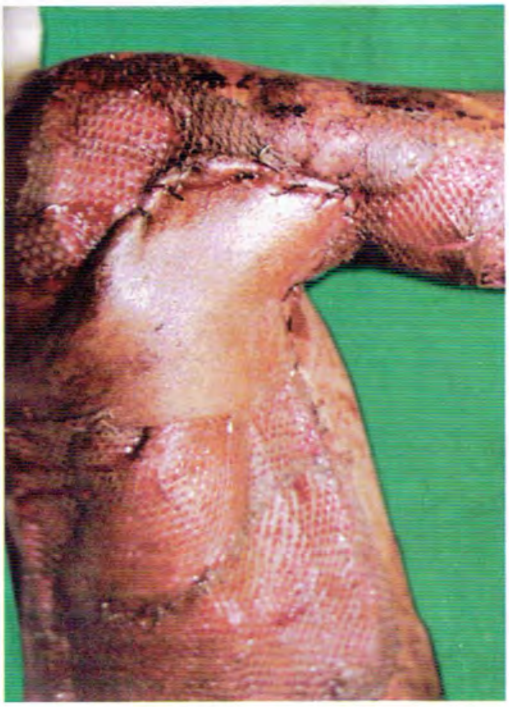


Fig. 21.45B: Cover over exposed vessels by latissimus dorsi musculocutaneous flap

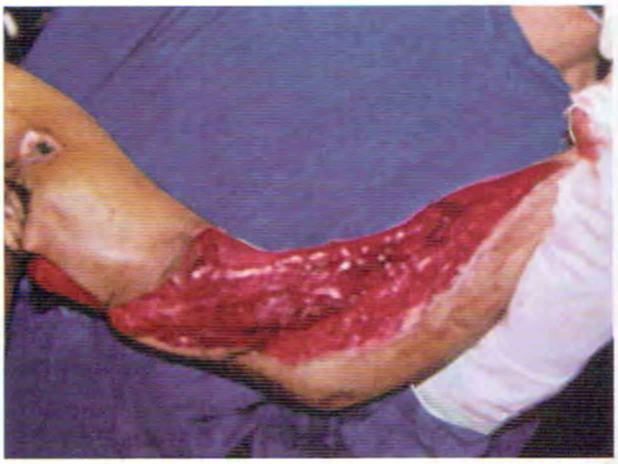


Fig. 21.46A: Reconstruction to save the involved neurovascular bundle in elbow by providing muscle cover (Latissimus dorsi muscle)

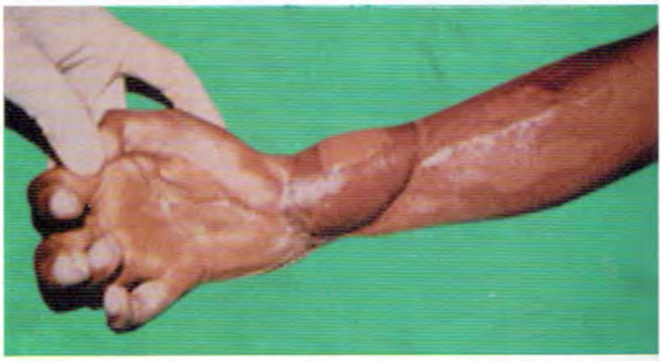


Fig. 21.46B: Reconstruction of hand after electrical burn – functional gracilis muscle transfer for finger flexion with groin flap cover over exposed vessels and tendons in wrist

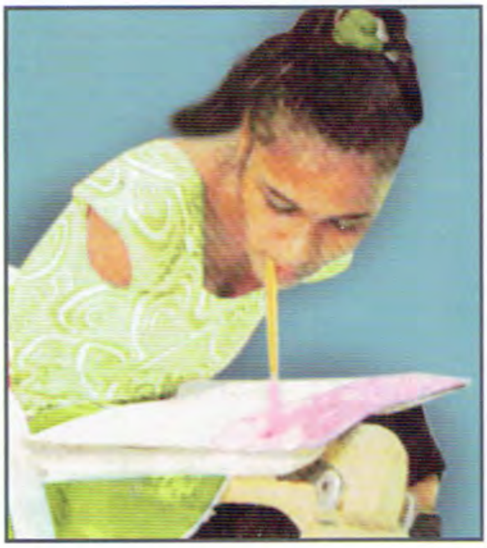


Fig. 21.47: A girl, who lost all her four limbs after 11,000-volt power line fell on her, paints with her mouth

rehabilitation of the patient, both physical/occupational and psychological, particularly if patient has undergone amputations, is very important in order to successfully rehabilitate the patient (Fig. 21.47).

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Chapter 22

Industrial and Chemical Burn

Mamta Sikr

INTRODUCTION

Industrial burns are similar to any other burns. The only major difference is labor and union problems and one must be fully aware of the labor laws and compensation. Also emphasis on safety measures is a must as the treatment of burn is a painful and prolonged process and also involves a lot of expenditure. Besides the loss to the company in terms of substantial man power loss, the cost of treatment and subsequent compensation involved puts a tremendous burden on the company. Any type of burn can occur depending on the type of industry the worker is employed with.

EPIDEMIOLOGY

The exact statistics of burn and cost of treatment is not available in India. In America a fatal injury occurs every 103 minutes and a disabling work place injury occurs every 8 seconds. Work place injuries cost Americans $ 125 billion in a year that's equivalent to nearly triple the combined profit reported by the top 5 fortune 500 companies in the same year. Work related burns account for 20-25%[1] of all serious burns. In India approximately 1 million burns occur every year. Percentage of industrial burns is not exactly known in India. However, in a steel industry like ours the work related burns which need admission account for 10-15% of total burn admissions.[2]

Except major disasters which occur once in a while, these burns are usually not very extensive and mortality is accordingly very low. Most of the work related burns are seen in welders where flash burns and explosions are the most common injury producing events. Other common
of injury is contact with hot metal (sometime molten m
These burns are very deep and cause severe morbidit
are rarely fatal.

Chemical burns again are quite frequently repor
work place but they are rarely fatal. The greatest ri
phenol injury is associated with manufacturing of
fertilizer, plastics and explosive and in hospitals wher
used as a disinfectant. Workers involved with glass et
process, petroleum refining and air conditioner cleani
exposed to the risk of hydrofluoric acid injury.

Anhydrous ammonia injury commonly occu
agricultural workers. White phosphorus and mustar
injuries mostly inflict military personnel.[3] Risk of injur
to strong acids occurs in persons who are involved in p
process and fertilizer manufactures. The greatest ri
strong alkali injury in the work place is associated
soap manufacturing.

Flame burns are less common in factories; usually
in restaurant workers but rarely serious to warrant ho
admissions.

Electrical current injuries are very common in lin
working on high tension wires. They usually present
very deep burns and even gangrene leading to mu
amputations. They are commonly associated with in
like head injury or spinal injuries.

Electric flash burns are usually seen in workers
are checking transformers or electrical switch board

Employees of utility companies, electric
construction workers (particularly those working
cranes), farm workers moving irrigation pipe, oil

workers, truck drivers and individuals installing antennae are at great risk of high voltage electric injury.[4] Hot tar burn is common in workers involved in building and road construction. Cement burns (due to lime in it) though reported in Western literature are very rarely seen in India.

Acid burns in Indian subcontinent are usually homicidal and only few cases are accidental. Accidents are usually domestic because of improper storage of chemicals used for cleaning toilets and drains. Work related acid burns are seen very often in jewelers who use sulfuric acid for cleaning purpose. Phenol burns are commonly seen in hospitals where it is used for carbolization of wards and operation theaters.[5-8]

PATHOPHYSIOLOGY

Pathophysiology of thermal burns and electrical burn is discussed in detail in chapters dealing with them. Contact burns with hot metal are thermal burns and so pathophysiology remains same. Here, we will only dwell upon the chemical burns. There are similarities between chemical and thermal burns, namely, both involve protein denaturing and produce wounds which look superficially alike. On the other hand, important differences do occur. Thermal burns are typically produced by very short term exposure to intense heat which is relatively quickly stopped.[5] Chemical injuries are more likely produced by longer exposure to chemical and this exposure may still be continuing in an emergency room. In thermal injuries, there is a rapid coagulation of protein due to irreversible cross-linking reaction, whereas with chemical burn the protein destruction is continued by other mechanisms. The commonest mechanism is hydrolysis which is driven by pH disturbances. Thus, there is an additional problem of restoring tissue pH in order to stop cellular damage. These mechanisms may continue so long as traces of the offending agent are present, especially in the deeper layers. The degree of tissue damage as well as the level of systemic toxicity is determined by the chemical nature of the agent, its concentration, the duration of skin contact and the mechanism of action. Chemical injuries are classified either by the mechanism of action on skin or by the chemical class of the offending agent.

MECHANISM OF ACTION

With the exception of concentrated or fuming sulfuric acids, thermal injury rarely plays more than a minor role in chemical injury. The main mechanisms of action on living tissue can be placed into six categories. The cells may be damaged by:

1. Reduction
2. Oxidation
3. Corrosion
4. Protoplasmic poisons
5. Desiccation or
6. Vesicant activity

Reduction: Protein denaturation is caused by the reduction of the amide linkages following exposure to reducing agents. Heat may also be a product of a chemical reaction, thereby causing a mixed picture. Reducing agents are rather rare and highly reactive chemicals, e.g. Alkyl mercuric compounds, diborane, lithium, aluminum hydride and other metallic hydrides.

Oxidation: Some agents cause damage by inserting oxygen, sulfur or halogen (most often chlorine) atom to otherwise viable body proteins. In general, this destroys a protein's ability to function. Sometimes, the oxidation product can have very untoward effects as it becomes a competitor for active metabolic sites. Common examples are sodium hypochlorite, potassium permanganate, all peroxides and chromic acid.

Corrosive agent: Some agents corrode the skin and cause massive protein denaturation. As a class they act in a variety of ways but they tend to produce a soft eschar which may progress to shallow indolent ulceration. Phenols are the commonest corrosive agents encountered. The hydroxides of sodium, potassium, ammonium and calcium follow as close second.

Protoplasmic poisons: Some poisons produce their effects by causing the formation of esters with proteins or by the binding or inhibition of the inorganic ions which are necessary for normal healthy cellular function. Ester formers are typified by formic and acetic acids while metabolic competitors or inhibitors include oxalic and hydrofluoric acids.

Desiccants: The damage by desiccants is caused by water being extracted from tissues by hygroscopic agents. The damage is often exacerbated by heat production as these reactions are usually exothermic. Concentrated or fuming sulfuric acid is the best known desiccant.

Vesicants: These are characterized by their ability to produce cutaneous blisters and are potent chemical warfare agents. They include mustards, arsenicals and halogenated oximes. Mustards are alkylating agents and the damage to

DNA renders them toxic to proliferating cells. Following death of the basal cells proteases are released from lysosomes causing the epidermis to separate from the dermis. The result is a blister with the basement membrane on its dermal side.[10]

Chemical Classes

The chemical class of a noxious agent is based on the chemical reactions which the agent is prone to initiate. Acidity/alkalinity or the ability to influence pH is one of the most important characteristics of all injurious chemicals and one which must be considered in effective wound management. It should also be noted that concentration plays an important role in the reactivity of a chemical. In general, concentrated solution (>70% wt) are more viscous, more reactive, more hygroscopic and liberate more heat during lavage or neutralization. The strength of an acid is defined by how easily it gives up the proton; the strength of a base is determined by how avidly it binds the proton. The strength of acids and bases is defined by using the pH scale, which ranges from 1-14 and is logarithmic. A strong acid has a pH of 1, and a strong base has a pH of 14. A pH of 7 is neutral.

The severity of a chemical burn is related to a number of factors, including the pH of the agent, the concentration of the agent, the length of the contact time, the volume of the offending agent and the physical form of the agent. The ingestion of solid pellets of alkaline substances results in prolonged contact time in the stomach, thus, more severe burns. In addition, concentrated forms of some acids and bases generate significant heat when diluted, resulting in thermal and caustic injury. [11]

The long-term effect of caustic dermal burns is scarring and depending on the site of the burn, scarring can be significant. Ocular burns can result in opacification of the cornea and complete loss of vision (Fig. 22.1). Esophageal and gastric burns can result in stricture formation.

Acids

Acids are proton donors and so they release hydrogen ions and reduce pH from a neutral 7 down to values as low as 0. Hydrogen ions are reactive and protonate any amine or carboxylic site. This causes a protein structure to collapse. After sometime, hydrogen ions catalyze protein hydrolysis into constituent amino acids. Most acids produce a coagulation necrosis by denaturing proteins, forming a coagulum (e.g., eschar) that limits the penetration of the acid. Mineral acids may also have other effects as seen in concentrated sulfuric acid injuries where there is generation and desiccation, producing a mixed ir Phosphoric acid for instance, can produce a similar n injury by chelating calcium as well. Hydrofluoric a somewhat different from other acids in that it produ liquefaction necrosis.

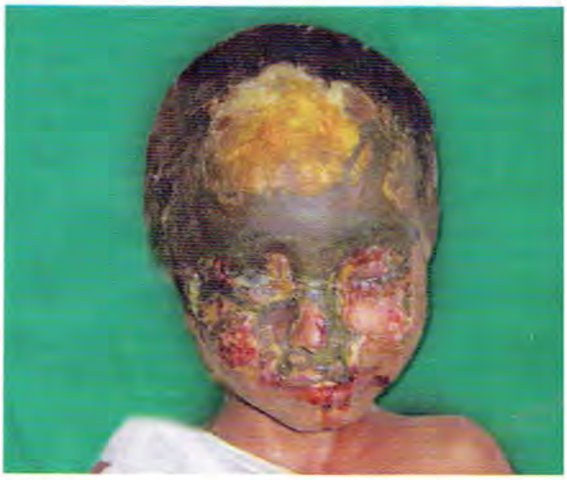

Fig. 22.1: Deep acid burns of face and scalp in a 4-year-old involving both eyes with complete loss of vision

Bases

Bases are proton acceptors. The major types to cause i are mineral alkalis. Bases strip hydrogen ions protonated amine groups and carboxylic groups. This c protein structure to collapse and in common with bases catalyse protein hydrolysis. Bases can also ca mixed injury by forming salts and soaps, causing t destruction as seen in the corrosive group. Bases typi produce a more severe injury known as liquefa necrosis. This involves denaturing of proteins as w saponification of fats, which does not limit tissue penetr Ammonia, the hydroxides of sodium and potassiur lime are the most common agents of this type to injury.

A large number of industrial and commercial pro contain potentially toxic concentrations of acids, bas other chemicals that can cause burns. Some of the common products are listed as follows:

Acids

- Sulfuric acid is commonly used in toilet bowl clea drain cleaners, metal cleaners, automobile battery munitions and fertilizer manufacturing. Concentra range from 8% acid to almost pure acid. The concentrated acid is very viscous and denser than water. It also generates significant heat when diluted. These attributes make sulfuric acid an effective drain cleaner. Concentrated sulfuric acid is hygroscopic. Thus, it produces dermal injuries by dehydration, thermal injury and chemical injury. It rapidly produces carbon from glucose in the tissues and thus typically produces a brownish black slough (Figs 22.2 to 22.4).

- Nitric acid is commonly used in engraving, metal refining, electroplating, and fertilizer manufacturing. It causes a typical orange yellow discoloration of the skin due to xanthoprotein reaction.
- Hydrochloric acid is commonly used in toilet bowl cleaners, metal cleaners, soldering fluxes, dye manufacturing, metal refining, plumbing applications, swimming pool cleaners and laboratory chemicals. Concentrations range from 5-44%. Hydrochloric acid is also known as muriatic acid and it produces yellow brown discoloration of skin (Fig. 22.5).
- Hydrofluoric acid is commonly used in rust removers, tire cleaners, tile cleaners, glass etching, dental work, tanning, semiconductors, refrigerant and fertilizer manufacturing and petroleum refining. This is actually a weak acid and in dilute form, it does not cause immediate burning and pain on contact..
- Phosphoric acid is commonly used in metal cleaners, rust proofing, disinfectants, detergents and fertilizer manufacturing.
- Acetic acid is commonly used in printing, dyes, rayon and hat manufacturing, disinfectants and hair wave neutralizers. Vinegar is dilute acetic acid.
- Formic acid is commonly used in airplane glue, tanning and cellulose manufacturing.
- Chloroacetic acids:
  - Monochloroacetic acid is used in the production of carboxymethylcellulose, phenoxyacetates, pigments and some drugs. It has significant systemic toxicity because it enters and blocks the tricarboxylic acid cycle, inhibiting cellular respiration. It is highly corrosive.
  - Dichloroacetic acid is used in manufacturing chemicals. It is a weaker acid than trichloroacetic acid and it does not inhibit cellular respiration.
  - Trichloroacetic acid is used in laboratories and in chemical manufacturing. It is highly corrosive and "fixes" tissues it contacts. It does not inhibit cellular respiration.

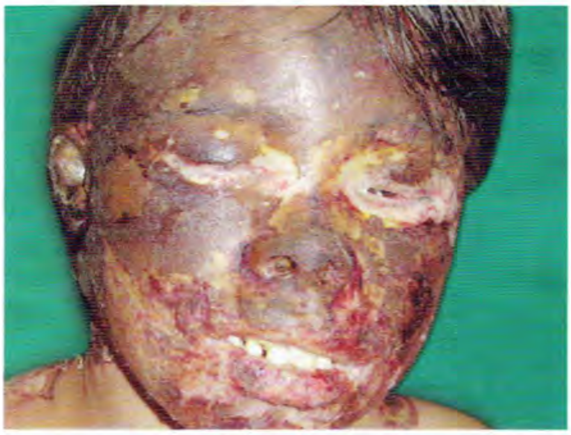

Fig. 22.2: Case of homicidal acid burns with superficial and deep dermal burns of face due to sulfuric acid showing brownish discoloration of skin and total loss of right ear

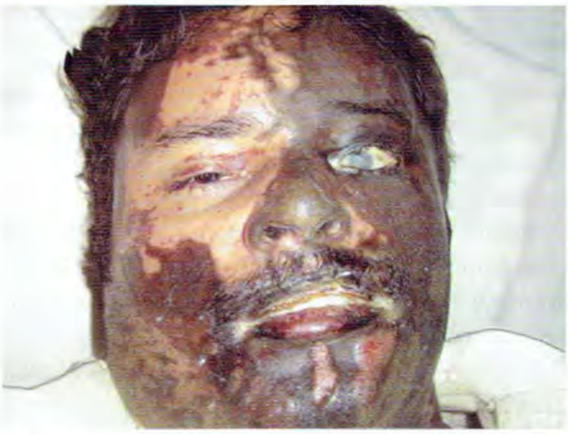

Fig. 22.3: Case of homicidal acid burns with brownish color eschar and involvement of left eye

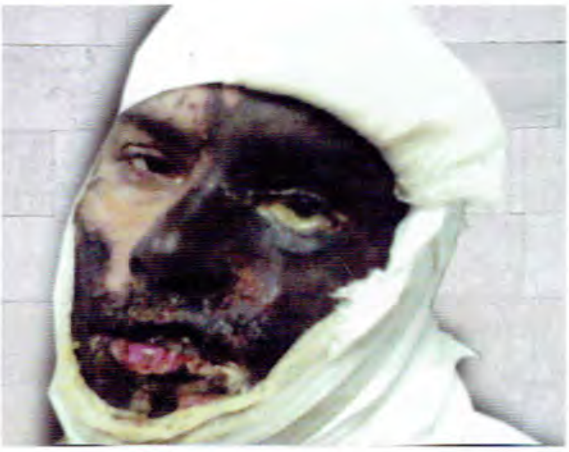

Fig. 22.4: Full thickness burns due to concentrated sulfuric acid with discoloration of sclera and black eschar on face

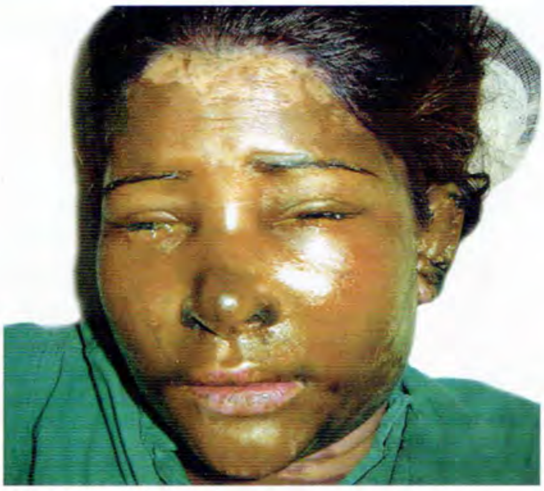

Fig. 22.5: Case of homicidal acid burns due to hydrochloric acid showing yellowish brown discoloration of skin

Bases

- Sodium hydroxide and potassium hydroxide are used in drain cleaners, oven cleaners, Clinitest tablets and denture cleaners. They are extremely corrosive. Clinitest tablets contain 45-50% sodium hydroxide (NaOH) or potassium hydroxide (KOH). Solid or concentrated NaOH or KOH is denser than water and generates significant heat when diluted. Both the heat generated and the alkalinity contributes to burns (Figs 22.6A to C).
- Calcium hydroxide is also known as slaked lime. It is used in mortar, plaster and cement. It is not as caustic as NaOH, KOH or calcium oxide.
- Sodium and calcium hypochlorite are common ingredients in household bleach and pool chlorinating solution. Pool chlorinators also contain NaOH and have a pH around 13.5, making them very caustic. Household bleach has a pH around 11 and is much less corrosive.
- Calcium oxide, also known as lime, is the caustic ingredient in cement. It generates heat when diluted with water and can produce a thermal or caustic burn (Fig. 22.7). Lime is very often used in our country for removal of moles and warts..
- Ammonia is used in cleaners and detergents. The dilute form is not very corrosive. Gaseous anhydrous ammonia is used in a number of industrial applications, particularly in fertilizer manufacturing. It is very hygroscopic (has a high affinity for water). It produces injury by desiccation and heat of dilution in addition to causing a chemical burn. It can cause severe skin burns as well as pulmonary injury.

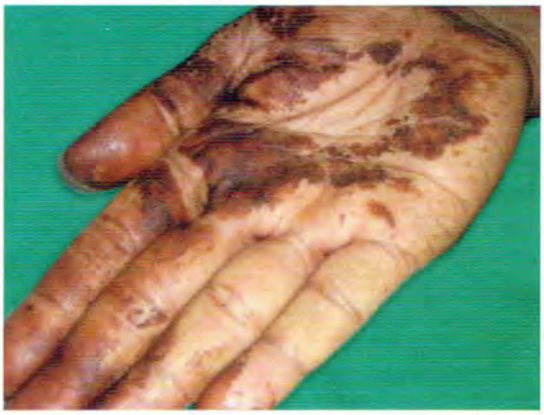

Fig. 22.6A: Accidental alkali burns of hand while using alkali i laboratory–Palmar aspect

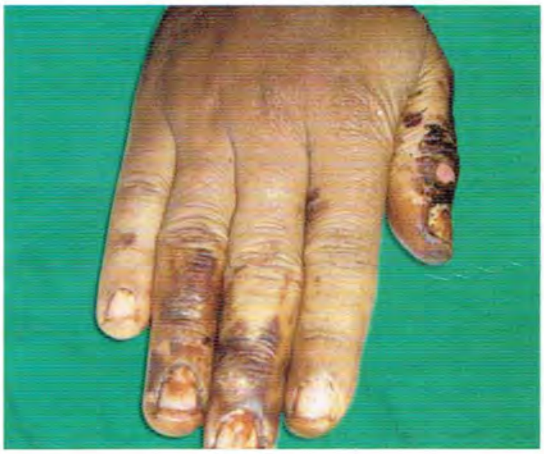

Fig. 22.6B: Same patient's hand as in Figure 22.6A–Dorsal asp

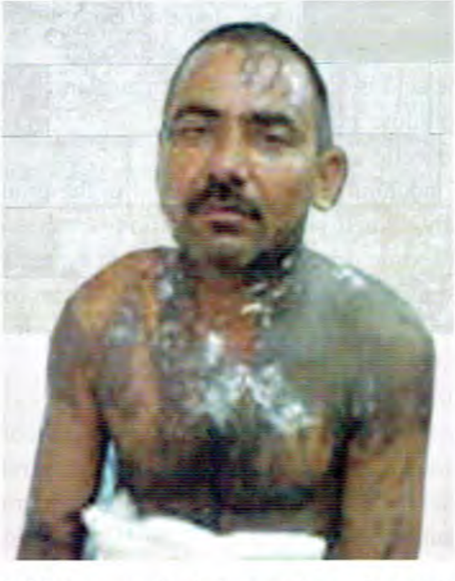

Fig. 22.6C: Patient with alkali burns due to Drainex(NaOH)

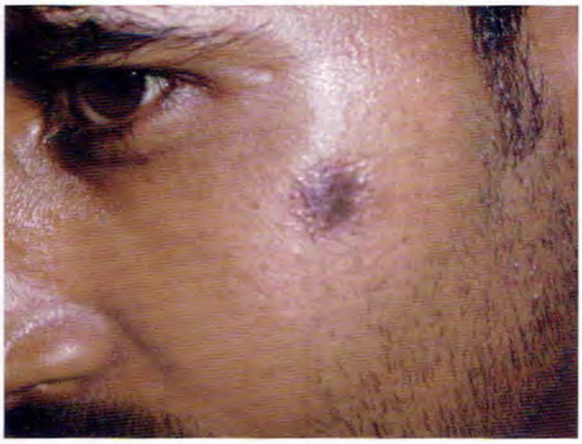

Fig. 22.7: Lime burn of face (lime used for 'treating' a mole)

- Phosphates are commonly used in many types of household detergents and cleaners. Substances include tribasic potassium phosphate, trisodium phosphate and sodium tripolyphosphates.
- Silicates include sodium silicate and sodium metasilicate. They are used to replace phosphates in detergents. Dishwashing detergents are alkaline, primarily to builders such as silicates and carbonates. They are moderately corrosive.
- Sodium carbonate is used in detergents. It is moderately alkaline, depending on the concentration.
- Lithium hydride is used to absorb carbon dioxide in space technology applications. It vigorously reacts with water to generate hydrogen and lithium hydroxide. It can produce thermal and alkaline burns.

Organic Compounds

A wide range of organic compounds damage protein by direct chemical reactions and heat production. They may also act as solvents and disrupt the protein structure. Such agents include phenol and petroleum based compounds.

- Phenol, also known as carbolic acid, is a weak organic acid used in the manufacture of resins, plastics, pharmaceuticals and disinfectants.
- Cresols are dihydroxybenzenes that are used as wood preservatives, de-greasing agents and chemical intermediates. These substances are very irritating to the skin and can be absorbed through the skin to produce systemic toxicity.

Inorganic Agents

Apart from the mineral acid and alkalis referred to above, highly reactive elements damage skin by direct binding and salt formation as well as heat production. Sodium, phosphorus, lithium and chlorine are excellent examples (Figs 22.8 and 22.9). Some salts especially fluoride can also cause damage.

Oxidants

- *Bleaches:* Chlorates are the primary chemicals used as bleaches in the United States. Household bleach is alkaline with a pH of 11-12, but it is dilute enough to be minimally irritating to the skin. More concentrated, industrial strength chlorates may be more damaging to the skin.

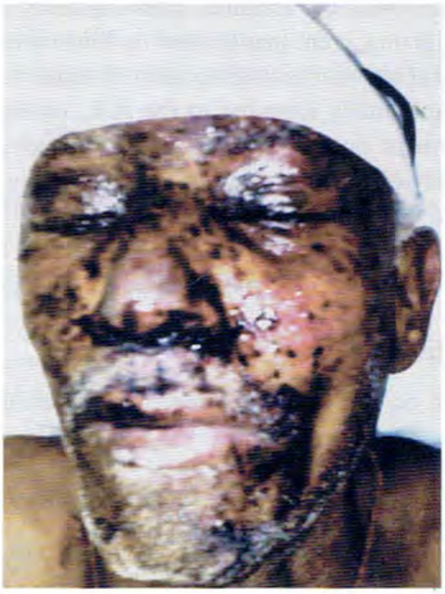

Fig. 22.8: Burns of face due to blast from a mixture of sodium and potassium that the patient was grinding

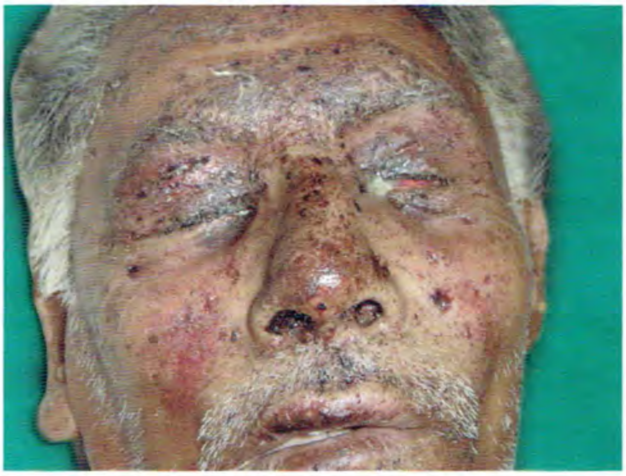

Fig. 22.9: Phosphorus burns of face due to accidental bursting of fire cracker

- *Peroxides:* Household-grade hydrogen peroxide (3%) produces minimal-to-no skin irritation. Concentrations of 10% may cause paresthesias and blanching of the skin. Concentrations of 35% or more cause immediate blistering.
- *Chromates:* Potassium dichromate and chromic acid are common industrial chemicals used in tanning, waterproofing fabrics, corrosion inhibitor, painting and printing and they are also used as an oxidizing agent in chemical reactions. Chromates can result in severe skin burns and subsequent systemic toxicity, including renal failure.
- *Manganates:* Potassium permanganate is a strong oxidizing agent that is used in dilute solutions as a disinfectant or sanitizing agent. In dilute solutions, it is minimally irritating to the skin. In concentrated form or pure crystals, it can cause severe burns, ulcerations and systemic toxicity (Figs 22.10A and B).
- Other common **Corrosives** which have been seen to cause burns are concentrated Savlon (Fig. 22.11).

MANAGEMENT

Starts with on site care and transfer to a specialized burn unit.

On Site Emergency Care of Burn Victims

Thermal burns caused by flame, steam, hot liquid or contact with a hot surface before the medical help arrives:

1. Stop the burning processes by using low pressure water
2. Provide first aid measures as needed (CPR etc)
3. Control bleeding (Burns do not bleed and if it is present there is another cause)
4. Have someone call for emergency medical assistance
5. Keep the victim warm using blanket
6. Stay with victim until help arrives

For tar burn cool the tar with water. Do not try to remove the tar at the accident site.

Electric burn: Caused by contact with electric current. Before medical help arrives:

1. If the victim is being held by the source, turn the current off or use a non-conductive item (like wooden stick) to knock the victim away from the source before attempting rescue.
2. If clothing has got ignited use low pressure water to extinguish the flame.
3. Provide first aid measures and CPR as indicated.[12]

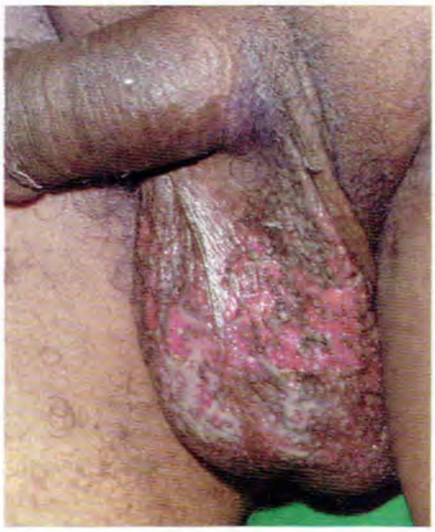

Fig. 22.10A: Perineal burns due to potassium permanganate used for sitz bath

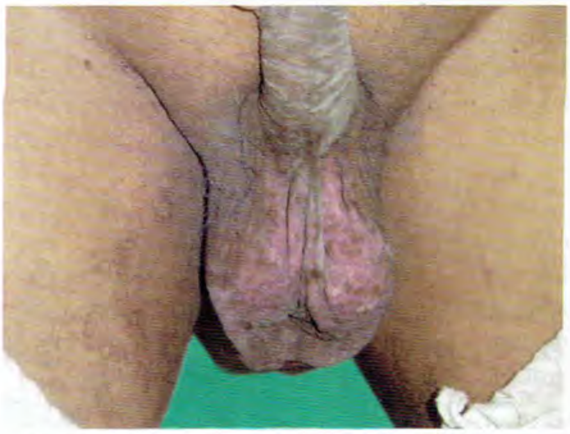

Fig. 22.10B: Same patient after complete healing of wounds in 2 weeks

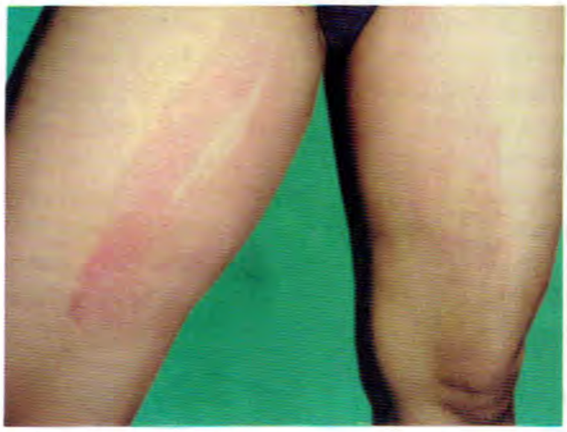

Fig. 22.11: Superficial burns caused by Savlon cleaning during dilatation and curettage operation in a female

Chemical burn: Caused by contact with a hazardous chemical. Before medical help arrives:

1. If in the powder form brush off as much as possible before using water[13]
2. Immediately use low pressure water (safety shower) to wash off the chemical [14]
3. Remove contaminated clothing, jewelry and contact lens while under water
4. Make the patient stay in shower for at least 30 minute to 1 hour before leaving for the hospital[15]
5. Take the MSDS sheet for the chemical to the hospital with the person
6. Do not use neutralizing agent.

If the eyes are involved:

1. Flush the eyes thoroughly using eye wash station
2. Hold the eyelid open while flushing and remove contact lenses while flushing with water
3. Do not use neutralizing agent

Once the Patient Reaches the Hospital

In the emergency take the history preferably from patient, if not possible, the accompanying person(s) can give the history. It is important to know the mode of burn and any associated injuries so that treatment can be instituted accordingly.

History

- Type of burn
  - Thermal
    - Scald
    - Flame
    - Contact
    - Flash
  - Non thermal
    - Electric
    - Chemical
    - Radiation
- Duration of exposure or contact with hot object
- In case of chemical burns concentration of the offending agent should also be known, the type of chemical (acid, base etc) and whether it was ingested or only dermal exposure should also be known.
- In case of electric burn one should know whether it was electric flash or high or low voltage contact electric burn
- Next important thing to know is whether patient had any fall from a height or whether any heavy object has fallen on the patient (common in case of steel industry) in case of hot furnace blast in which patient sustains flame burns as well as soft tissue and bony injury as well (Figs 22.12A to C).
- Other important point is to know whether water has been used to extinguish the flames or cool down the skin surface or to wash off the chemical from the skin surface as it definitely reduces the depth of burn.

After taking history, patient should be thoroughly examined to know:

1. The airway and circulation status
2. Extent and depth of burn
3. Presence of any head injury or spinal injury which takes priority over burn treatment
4. Any ocular exposure (specially in chemical or electric flash burn)
5. Presence of any long bone fracture.

Treatment in Emergency Room

1. Ensure airway patency and breathing
2. Establish good intravenous line and start IV infusion with ringer lactate
3. Intravenous analgesics and/or sedation (if head injury is ruled out)
4. Stabilize fracture(s) with splint
5. If chemical burn, then thorough washing with water must be done for at least 30 minutes to 1 hour and further confirmation for whether adequate washing has been done is by litmus test.
6. CT scan for head injury and spinal injury should be done
7. ECG should be done if there is history of electric current injury

After this patient should be transferred to burn ward.

In the burn ward the patient should be examined in detail. History is taken again in detail. Any history of an associated illness like diabetes mellitus, hypertension, ischemic heart disease, etc. must be taken as this affects the prognosis. In female patients history of pregnancy and lactation must be taken. Physical examination should be done in detail again to know the extent and depth of burn and any other associated injury.

If a good intravenous access has not been already set up in emergency room, it must be started now.

Patient should be catheterized. Analgesics and/or sedatives should be given.

If patient is having any associated inhalation injury, bronchodilators and oxygen by mask should be given. If these measures do not suffice, endotracheal intubation or

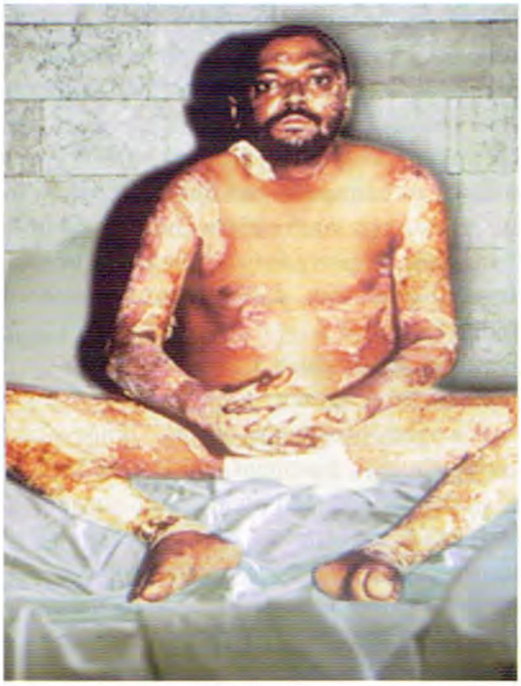

Fig. 22.12A: 80% flame burns sustained during a hot furnace blast

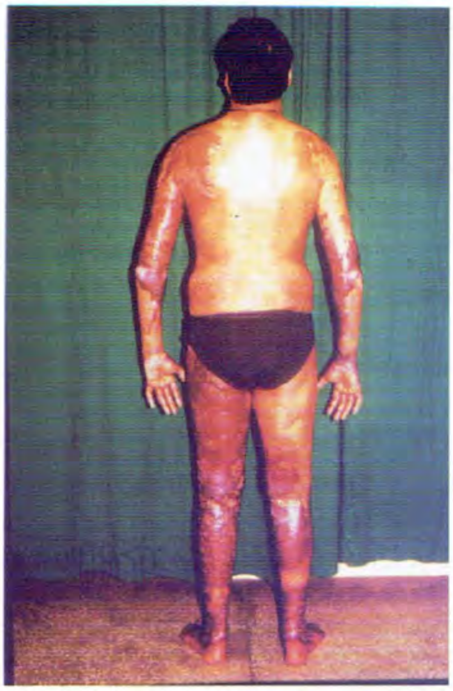

Fig. 22.12C: Same patient from back showing the hypertrophic scarring in lower limbs and upper limbs

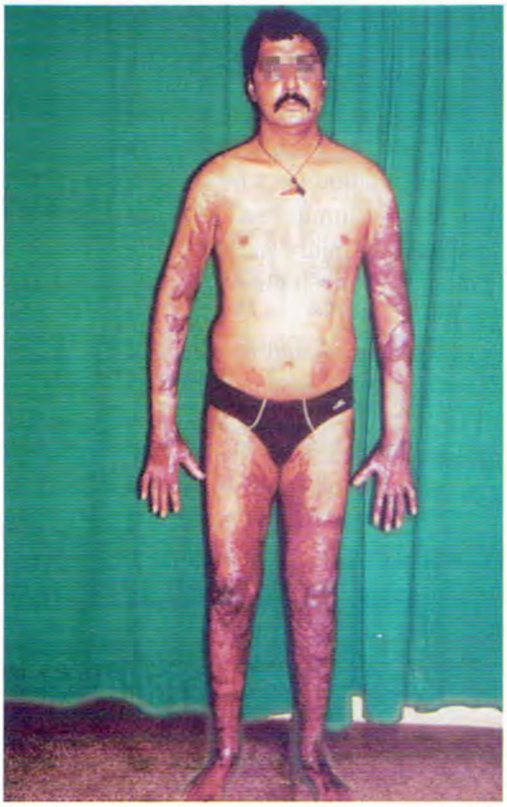

Fig. 22.12B: Same patient after complete healing from front

tracheostomy can be done. Intravenous fluids should be given using the Parkland formula or modified Brooke formula aiming to keep the urine output at 0.5-1ml/hour/kg. In electric burn more intravenous fluids (as much as 6-7 ml/kg/% burn) are required to flush out the pigments released by damage to RBC or muscles. If need be, mannitol can be given to increase urine output.

Once the patient is stabilized with sufficient intravenous fluids and analgesics and the urine output is satisfactory, the wound should now be dressed and the investigations are sent.

Investigations

Routine: Complete hemogram, biochemistry (urea, sugar, creatinine, electrolytes, etc.) serum protein and albumin level and sometimes serum transferrin level, if available. In case of electric burn, samples for serum creatinine phosphokinase and lactic dehydrogenase should also be sent.

If required, serum bilirubin, SGOT, SGPT, alkaline phosphatase etc. should be sent specially in chemical burns.

In tribal areas and in certain communities like Sahu in Chhattisgarh, sickling test must be done routinely and if required complete Hb electrophoresis should be done.

Blood grouping should be done routinely.

ECG should be done routinely in all electric burn patients and in elderly patients.

X-ray chest should be done to rule out any hemothorax or pneumothorax in electric burn patients or in any other patient, if required.

Radiography of spine and long bones should be done if there is history of fall.[16]

Computerized tomography and radiography of skull should be done again to rule out head injury in suspected cases.[12]

Gastrografin contrast studies may be indicated for a suspected perforated viscera.

Wound Management

In case of thermal burns (scald, flame, contact, flash, etc.) wound is first washed thoroughly with any soapy solution (surgiscrub or savlon) to remove all dirt or grease, etc. All blisters are punctured, large blisters should be debrided. The wound should be washed with saline or distilled water.

Second degree superficial and deep dermal burn wounds are dressed with antimicrobial creams, nonadherent and absorbent dressings. Skin substitutes may be used depending on their availability in a particular hospital.

Various biological dressings available in India are amniotic membrane, collagen, etc. Integra[17] which is a double layered biological dressing (outer Silastic epidermis and inner artificial dermis) is available but very costly. Other skin substitutes like porcine skin and cadaver skin are not easily available for use in India.

Biological dressings reduce the number of dressings thereby reducing the work load on the burn team. Other important advantages of biological dressings are that they reduce evaporative water loss from denuded body surface thereby reducing fluid requirement, reduce pain as the exposed nerve endings are covered and also reduce the need for antibiotics by preventing bacterial invasion of wound.

Sometimes, in second degree deep dermal burns the biological dressing is rejected. Still the wound healing is enhanced as initial prevention of wound desiccation (because of evaporative loss) and bacterial invasion prevents conversion of second degree deep dermal wound into third degree burn. After the initial biological dressing wound is opened after 48 hours and reviewed. If the dressing is adherent and patient is stable he/she can be discharged with advice to come for out patient treatment.

If burns are very extensive, especially in circumferential burns, biological dressings become too costly and we opt for conventional dressings. Again for the same reason because the biological dressing does not adhere it is not used for third degree burns.

We go for conventional dressing in extensive circumferential burns and in third degree burns. After cleaning with surgiscrub and saline a final wash with 5% povidone iodine lotion is done (optional) and then dressing is done with 1% silver sulfadiazine ointment. The author is using silver sulfadiazine ointment mixed with 30% Aloe vera juice for dressing in their setup. It is known that silver sulfadiazine inhibits the wound epithelization [18]and aloe vera enhances epithelization by increasing wound circulation and enhancing collagen formation.[19]

Aloe vera acts as an antimicrobial also as it prevents bacterial growth. Ointment like mafenide acetate is very good for burn wound as it has very good penetration of eschar but it is not available in India and is also very toxic.[20]

Other ointments like povidone iodine, nitrofurazone, Neosporin, etc. are also in use but they are better used for small wounds as their antimicrobial spectrum is very limited. For extensive burns, silver sulfadiazine is the best.[21]

In third degree circumferential burns escharotomy should be done once the patient is adequately resuscitated as distal circulation may be compromised or in case of burns of the trunk, respiration may be compromised.

In electrical burns fasciotomy must be done immediately.[12] Other resuscitative measures are same as for thermal burns.

Chemical Burns

The management of chemical burns differs a little from other burns.[22-24]

Management Guidelines for Chemical Burns

1. Evaluate ABC (Airway, Breathing, Circulation)
2. Start decontamination:
    A. Ensure medical personnel are protected
    B. Remove all clothing (undress to access)
    C. Wipe away or debride all solid chemical particles
    D. Inspect hair, nails and web spaces for collections of the chemical
    E. Remove caked compounds with green soap solution (liquid soap and 10% Isopropyl alcohol)
3. Obtain history
    A. Type and concentration of chemical
    B. Nature and duration of exposure
    C. Concomitant traumatic injuries

   D. Pre-existing medical illnesses
   E. Tetanus immunization and allergies
4. Physical examination
   A. Exposure by ingestion may lead to esophageal edema, stridor, use of accessory muscles, etc.
   B. Dermal exposure: Size, depth, location, circumferential burns, etc.
   C. Ocular exposure
   D. Visual acuity
   E. Presence of periorbital dermal lesion
5. In case the nature of the agent is not known, litmus paper test can be done to find out whether agent was acid or alkali (Fig. 22.13).
6. Start treatment with specific antidotes if applicable (only after initial thorough irrigation with water
7. Identify hospitalization criteria
8. Check effectiveness of irrigation, use pH/litmus paper; if the pH is not neutral, continue irrigating for an additional hour (Dilution is the solution to chemical pollution).

Caustic Ingestions

- Gastric emptying is contraindicated. Activated charcoal is not useful and may interfere with subsequent endoscopy. Dilution with milk or water is contraindicated if any degree of airway compromise is present. Milk may interfere with subsequent endoscopy. Water is benign. Some substances, such as drain cleaners containing sulfuric acid or sodium hydroxide, generate heat when diluted with water. Local areas of heat generation can be minimized by diluting with a moderate quantity of fluid (250-500 ml).

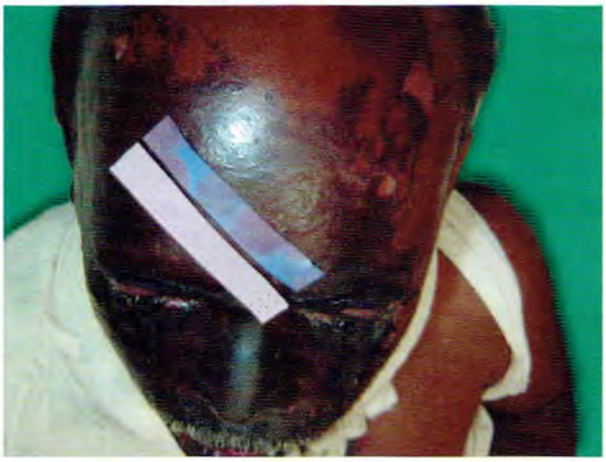

Fig. 22.13: Litmus paper test to identify the chemical (blue litmus turning red indicated acid and red paper turning blue indicating alkali)

- Do not attempt to neutralize the caustic agent. Neutralizing the caustic agent may generate excessive heat from the exothermic reaction of neutralization.

Endoscopy for Ingestions

- Perform esophagoscopy and gastroscopy on all patients with symptomatic ingestions and on patients who are asymptomatic but have a history of a significant ingestion of a substance with the potential to cause major injury.
- Findings on esophagoscopy do not correlate well with physical signs and symptoms. Of patients with esophageal injuries, 2-15% have no oral burns.
- Burn findings are classified as superficial, transmucosal, or transmural.
- Esophagoscopy findings are used to guide further treatment. The presence of full-thickness or circumferential burns is associated with future stricture formation.
  - The issue of whether to extend the endoscopic examination past the first site of injury is controversial.
  - Esophageal and gastric burns can result in stricture formation.

Ocular Exposures to Chemicals[25]

- The goal for decontamination should be to achieve a pH (of the eye wash) of at least 7.2, preferably 7.4.
- If pH paper is not available, an adequate guideline is decontamination with 1-2 L of irrigation fluid over 30-60 minutes.
- Ocular burns can result in opacification of the cornea and complete loss of vision (Fig. 22.14).

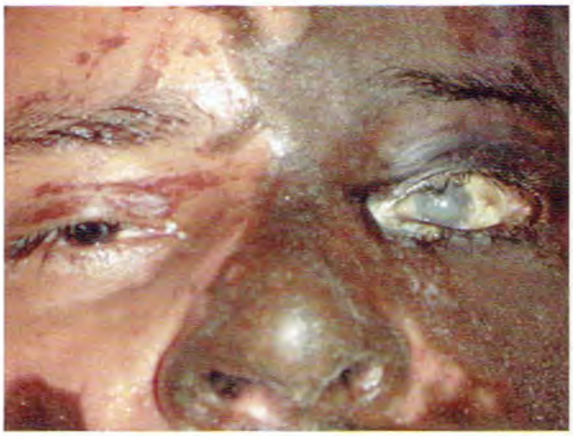

Fig. 22.14: Ocular chemical burns showing complete corneal opacification

- Indications for hospitalization of patients with chemical burns are almost same as other burns like:
  - High risk patients (concurrent illnesses)
  - Burns of hand, foot, face, eye or perineum
  - Burns of greater than 15% total body surface
  - Deep burns (second degree deep dermal or third degree)
  - Burns with substances that have systemic toxicity.

SPECIFIC AGENTS

In case of an industrial setting the causative agent is usually known. The following list of chemical is not definitive but it provides an inventory of the commonest agents to cause chemical burns.

Acetic Acid

This acid is a protoplasmic poison. Glacial acetic acid nearly 100% pure is the usual offender. Vinegar, a 5% solution of acetic acid, is usually benign unless scalding hot.

Alkali (Strong)

Multiple chemical agents are involved in caustic alkali burns. Lime, potassium hydroxide and sodium hydroxide are the most common agents causing chemical injury. Accidental injury occurs in infants and toddlers. The mechanism by which these alkali injuries are caused is due to three factors:

1. Saponification of fat causes fatty tissue to lose its function with increased damage due to heat reaction
2. Extraction of considerable water from cells cause damage because of an alkali's hygroscopic nature; and
3. Alkalis dissolve and unite with the proteins of the tissues to form alkaline proteinates, which are soluble and contain OH ions and these ions cause further chemical reactions which initiate deeper injury of the tissues.

As with other chemical burns alkalis are capable of deep penetration and can cause severe pain. For treatment it is necessary to remove the causative substance as quickly as possible by washing with large volumes of water. Washing is presumed to cause dilution and elimination of a chemical substance, attenuation of the chemical reaction and suppression of any raised tissue metabolism to have an anti-inflammatory action, to suppress the hygroscopic action and to return skin pH levels to normal. In lime burns the dry lime must be brushed away before washing in order to minimize the production of heat.

It is very difficult to determine the depth of tissue damage on admission to a hospital because chemical burns are different from thermal burns. Because the corrosive material blackens the skin, rapidly converting it to a firmly adherent, firm, dry eschar, a true assessment of the depth of an injury can only be made in retrospect as spontaneous healing or non-healing occurs.

Ocular damage is a common sequel to alkali injury. Damage is related to the pH: the higher the pH, the greater the damage to the eye, with the most significant injury occurring around pH 11-11.5. Alkali penetrates quickly and with little resistance, saponifies plasma membranes, denatures collagen and causes vascular thrombosis in the conjunctiva, the episclera and even in the anterior uvea. Very small volumes of alkali material can cause significant damage. Such damage may be made worse by repeated attempts to wipe off the chemical agent with the hands or inadequately washing the eyes. The policy is to treat with topical anesthetics followed by copious irrigation, induction of cycloplegia and mydriasis as soon as possible. Antibiotics such as gentamicin and chloramphenicol are effective. Topically cysteine, a collagenase inhibitor is applied every 2 hours in order to prevent perforation. Topical steroids are applied four times a day after complete epithelization has taken place.

Assorted Acids

Tungstic, picric, sulfosalicylic, trichloroacetic and cresylic acid are all strong protoplasmic poisons. They form firm eschars which may help spare underlying tissue from damage. Tannic acid has been used in the past as a treatment for thermal injury. These acids are absorbable and cause metabolic symptoms and are both renal and hepatic toxic. Most have pungent, suffocating odors and have wide industrial usage.

Cement

Cement is really an alkali but warrants a separate mention as it differs in many ways. When mixed with water, sand and gravel, it becomes more abrasive and is known as concrete. Injuries are not infrequently found in construction workers and do it yourself enthusiasts.[26] Cement (calcium oxide) penetrates clothing and reacts with sweat and an exothermic reaction takes place. The dry powder is very hygroscopic and causes a desiccation injury if not exposed to moisture. Hydrated calcium oxide becomes calcium hydroxide. An injury progresses as with a strong alkali. The critical substance responsible for skin damage is the hydroxyl ion.[27] A victim may not know until many hours later that a burn has been sustained as it is often a painless occurrence under clothing. Treatment consists of removal of contaminated clothing and washing the affected area with soap and water. Surgical excision is likely to be required as these injuries are often deeper than they appear.

Chromic Acid

A powerful oxidizing agent, chromic acid is used in the cleaning of other metals. The active chemical is chromic trioxide (CrO_3) in a solution of strong sulfuric acid. If a sample is examined it is a pungent viscid yellow liquid. Contact with skin causes protein coagulation, blister formation and ulceration. Ingestion leads to gastroenteritis which becomes severe very rapidly with vertigo, muscle cramps, peripheral vascular collapse and coma. This is followed by nephritis and is associated with glycosuria. The lethal dose is from 5 to 10 gm. Primary treatment of contact areas comprises water lavage to dilute the acid. Washing with a dilute solution of sodium hyposulfite followed by a rinsing with a buffered phosphate solution is the treatment of choice and should be available if this agent is in common use (70 gm monobasic potassium phosphate and 180 gm dibasic sodium phosphate to 850 ml of water). Systemic manifestations are to be treated with Dimercaprol 4 mg/kg by deep intramuscular injection every 4 hours for 2 days, followed by 2-4 mg/kg daily for about a week along with support for the failing organ system.

Formic Acid

Despite the common use of this organic acid for industrial de-scaling and as hay preservative[28], dermal injury is rare. All patients injured by formic acid should be hospitalized, if only for observation because cutaneous absorption from the injured area can cause metabolic acidosis, intravascular hemolysis, hemoglobinurea and renal failure as well as pulmonary complications, including ARDS. Vomiting and abdominal pain are usually common and although the cause is unknown, necrotizing pancreatitis has been described as a complication of formic acid ingestion. However, elevated serum amylase has not been reported in formic acid poisoning. Acidosis should be corrected with intravenous sodium bicarbonate. Minor hemolysis requires no treatment, but diuresis with mannitol is recommended where hemolysis occur after deep burns. Where extensive absorption of formic acid has occurred, hemodialysis should be considered. The wound has a greenish hue and is followed within 48 hours by gross edema and blister formation. Wounds tend to be deeper than they initially appear and are best treated by surgical excision.

Hydrocarbons

Petrol burns are seen in patients trapped under vehicles following road traffic accidents. The solvent properties of hydrocarbons promote cell membrane injury and dissolution of lipids, resulting in skin necrosis. Symptoms present with erythema and blistering. Burns tend to be superficial and heal spontaneously [29], grafting is usually not necessary although full thickness burns do occur. If absorbed systemically, toxicity can cause respiratory depression. If ignited deep thermal injury is common.

Exposure to gasoline may be complicated by the leading agents that are used to improve the performance characteristics. Significant lead absorption and subsequent neurological damage may ensue from gasoline exposure. Tetraethyl lead is more toxic than elemental lead in this regard.

Common complications from the contact effects of gasoline and other light petroleum products such as kerosene include toxic effects from inhalation and ingestion. In addition to the skin burn, the patient often may have serious multi-system effects, including organ system failure. Inhalation or absorption of gasoline produces symptoms similar to those of ethanol in descaling, including ataxia, mental confusion and blurred speech. High concentrations of gasoline vapors may lead to coma and death. Once far more common, the gasoline snifter is much less frequently found now.

Cardiovascular complications include sudden death when inhalation is followed by strenuous exercise or stressful situations. Spontaneous and severe cardiac arrhythmias are the causes of death in these cases. Catecholamine release appears to increase the toxic potential of the petroleum distillate.

Gasoline absorption has produced renal damage to include glomerulonephritis and renal tubular acidosis. The hydrocarbon hepatitis is secondary to vascular endothelial damage and is usually a reversible lesion without long term sequel.

Initial treatment of the gasoline immersion burn is to remove the clothing and cleanse the skin surfaces. Further emergency treatment of a petroleum distillate burn should include low pressure irrigation with water for at least 1 hour. The patient should be admitted to the hospital and observed for at least 24 hours. A monitored bed is appropriate for these patients. Serum lead levels should be obtained and treatment initiated if the lead level is greater than 30 to 40 nanogram per 100 ml. Dimer caprol (British Anti-lewisite) and calcium EDTA followed by penicillamine therapy for 15 to 20 days has proved efficacious in these cases.

Hydrofluoric Acid Burns

Hydrofluoric acid (HF) is a colorless liquid or gas that is manufactured by allowing sulfuric acid to react with fluorospar in heated ovens. HF was first manufactured by an English glassworker in about 1720.[21] It is produced as a gas, purified and then condensed in a liquid form. An aqueous solution will fume when the concentration is greater than about 50%. HF is a weak acid and is 1000 times less dissociated than hydrochloric acid. It means that HF can be absorbed or penetrate through the skin without dissociating into the constituent hydrogen and fluoride ions.

Hydrofluoric acid is used in glass and semiconductor industries as an etching agent, in the plastic industries in production of various plastic materials and in the nuclear industries as a solvent for uranium. Many rust removal agents contain hydrofluoric acid. Most hydrofluoric acid burns appear to come from either the semiconductor or the glass industry.

Hydrofluoric acid is very damaging to tissues even at low concentrations.[30] An HF solution of greater than 50% concentration produces immediate pain and tissue destruction. Solutions of less than 20% may cause burns delayed as long as 24 hours after the exposure while intermediate strength solutions can cause symptoms as long as 8 hours after exposure. The fluoride ions may enter the body through the skin or any mucous membrane, including the respiratory and gastrointestinal system.

The fluoride ion causes a breakdown in all calcium-dependent processes in the cell membrane. This destruction of the cell membrane causes liquefaction necrosis of the skin and subcutaneous tissues. The salts formed by HF are soluble and permeate through the tissues with continued destruction. The fluoride ion is also a cellular poison and causes cell death by blocking glycolysis.

As the salts continue to be bioactive, the fluoride ions must be inactivated. Without specific treatment, the injury is progressive and potentially fatal. Calcium and magnesium ions form insoluble salts with the fluoride ions. All other salts are soluble and dissociate completely allowing the continued diffusion of ions into the tissues.

$$\mathbf{HF = H^+ + F^-}\ \textbf{(Toxic)}$$

Immediately after the exposure, the area should be flooded with copious amounts of water at low pressures. While in the shower, the worker should remove all clothing and shoes to decrease the possibility of further contamination. After the area has been flooded with water for at least 30 minutes, the remaining fluoride ions can be fixed in insoluble salts with the application of topical magnesium oxide ointment or calcium chloride/calcium gluconate solutions. Of these, calcium holds the most promise for skin burns from HF.

$$\mathbf{Ca^{++} + F^- = CaF_2}\ \textbf{(Nontoxic)}$$

Some authorities also recommend the use of quaternary ammonia compounds such as Hibclens or Zephiran. These ammonia compounds have a dubious effect upon fluoride skin poisoning. They may also tend to irritate sensitive skin like that of the face.

Initial Presentation

The initial presentation is usually pain at the site of the exposure. Local erythema and edema may be present. The pain is uniformly described as excruciating, particularly after exposure to solutions of greater than 20% concentration. Also, the pain is often described as deep, throbbing or burning. Regional nerve blocks may be required for relief. Exposure to concentrated HF over as little area of skin as 2.5% of TBSA has produced fatal systemic fluoride poisoning.

As the injury progresses, erythema and edema progress to blanching. Bullae denote a more severe burn that requires aggressive treatment. A grossly necrotic area may develop with a subsequent sloughing of tissues and slow healing wound. In severe cases, bony injury may manifest and severe decalcification may be noted. Hydrofluoric acid burns have caused systemic hydrofluorosis with subsequent death.

Initial Treatment

If there is evidence of skin damage, even in the form of erythema, the skin has been penetrated by fluoride ions and these must be inactivated. It is probably appropriate and safe to assume that all spills are contaminated and that the patient will develop an injury, if not treated. Before proceeding, the provider must ensure that adequate protection against this agent is available and being used. Butyl rubber or nitryl gloves and apron are worn as a minimum precaution. Shoe covers decrease both "tracking" and contamination of the provider's shoes. All contaminated clothing and bedding should be placed in heavy plastic garbage bags and prominently labeled to ensure that there is no spill.

The area should be copiously flushed with running water while materials and further preparations are in progress. All bullae and vesicles should be aspirated to remove fluoride-containing fluids. Care must be taken not to spill the aspirate on any other part of the skin. The lesions must then be debrided.

Local Infiltrating Therapy

After debridement of the lesions, 10% calcium gluconate is injected into the burn to further bind the fluoride ions. About 0.5 ml of solution should be injected intradermally for every square centimeter of tissue damage.[31] The injection is extended about 0.5 cm into the margins of the burn to bind ions that may have already migrated there. To ease the pain of injection, a 27 or 30 gauge needle is used and the area slowly infiltrated. The calcium gluconate injection is usually effective in relieving the pain of the burn. Unfortunately, the volume of the calcium chloride is limited to only 0.5 ml per injection. This severely restricts the amount of calcium that can be injected therapeutically in one sitting. Recurrence of the pain is an indication of further fluoride ion migration and another injection is needed.

Intra-arterial Injection

Intra-arterial calcium may provide better tissue distribution and less tissue distention than injection into the burnt area. The arterial catheter should be placed in the distribution system, of course. Arterial injection of calcium may provide relief from burns to the finger tips without loss of either nail or distal finger. The calcium may be injected as either gluconate or chloride over four hours. If the patient complains of continued pain, additional calcium may be infused. Intravenous administration of calcium has not been effective as the concentration of calcium salts is limited by systemic calcium toxicity.

Eye Injuries

HF solution or vapor in the eye causes a more extensive damage than other acids in similar concentrations. The deeper penetration and liquefaction necrosis makes HF more destructive to the eye. Eye burns should be treated with immediate and copious irrigation of the eye immediately after exposure. If available, a morgan contact irrigating lens may be ideal for this. Eyelid spasm may be decreased by use of tetracaine or other ophthalmic anesthetic solutions. Following this irrigation, a 1% calcium gluconate solution in isotonic sodium chloride can be used as drops and instilled every 2-3 hours. Some workers feel that only irrigation with water, isotonic saline solution or magnesium chloride solution offer any therapeutic benefit. Other therapies that have been used on skin have been associated with increased rates of ocular injuries. Prompt ophthalmologist's consultation is essential.

Dressings

After the injection of calcium, the area may be covered with either calcium or magnesium oxide dressings. Magnesium oxide dressings are less expensive and easier to obtain but not as effective as calcium-based dressings. Calcium chloride soaks may also be used. Some authors recommend 2.5% calcium gluconate in a gel vehicle but further clinical trials are indicated before advocating this more costly therapy. A mixture of 2.5 to 5.0% calcium gluconate in KY jelly may be used as an inexpensive alternate.

The wound must be inspected frequently to determine if there is any further damage. Recurrence of pain is an indication for re-inspection and re-injection. A fluoride burn should always be re-inspected at 24-hour intervals, no matter how small or innocuous it seems.

Hydrofluoric acid can rapidly penetrate the nail bed and destroy the underlying nail bed and matrix. Infiltration of calcium ions into the subungual spaces is extremely painful and may cause vascular compromise in the restrictive space between nail and nail bed. When ungual areas are involved in a hydrofluoric acid burn, the affected nail must usually be excised under regional anesthesia. Removal of the nail allows regeneration after the injury.

Treatment that has been proposed in the past includes steroids and early surgical excision of the lesion. Steroids have not shown any beneficial effect and surgical therapy may be unnecessarily mutilating.

Complications

Skin burns with hydrofluoric acid may be associated with significant systemic absorption of fluoride ions. The fluoride ion can cause decalcification of bone and systemic hypocalcemia as a result of the formation of poorly soluble calcium fluoride complexes. This precipitous drop in serum calcium has been implicated in fluoride-induced sudden death. The mechanism of death is usually intractable ventricular fibrillation but this dysrhythmia does not respond to usual doses of intravenous calcium.[32] Several authors have reported use of massive doses of calcium salts with relief of the hypocalcemia. This profound hypocalcemia is often not associated with tetany.

Other authors feel that the leak of potassium from fluoride-damaged cells causes a fatal hyperkalemia. This hyperkalemia is mediated by calcium-dependent channels and has been blocked in dogs by quinidine. There are no reported trials of quinidine in fluoride toxic humans with hyperkalemia, hypocalcemia, and ventricular dysrhythmias. However, in view of the poor response to extant therapies, quinidine may be appropriate in these selected patients.

If there is any evidence of systemic fluorosis or the possibility of inhalation injury, the patient should be admitted to the hospital and observed for at least 24 hours. In these patients, monitoring of liver function studies, renal function studies, electrolytes and serial serum calcium levels in indicated.

Elemental Sodium, Potassium and Lithium Burns

Sodium, potassium and to a lesser extent lithium spontaneously ignite when in contact with water, including water vapor in the air or on the skin (Fig. 22.8). Using water to irrigate these burns only intensifies the combustion effects. When these metals combine with water, sodium, potassium and lithium hydroxides are formed. These are among the strongest of alkalies and capable of causing severe caustic burns.

To extinguish these burning metals, the oxygen and water must be removed from the area. A class D fire extinguisher or sand may be used to smother the fire. After the fire is extinguished, the area should be covered with oil, such as mineral oil or cooking oil to isolate the metal from water. Small metal pieces should be removed by debridement to healthy tissues. Embedded pieces must be removed surgically. Following debridement and ensuring that all pieces are removed, the area must be irrigated with water, using the same techniques as for an alkali burn.

Light Metals

Water is generally contraindicated for extinguishing burning light metal fragments because of the hydroxides and hydrogen gases that are produced when water is added to hot metals. Particularly injurious are burning fragments of elemental magnesium, which produces magnesium oxide. If the pieces of magnesium are not removed, the small ulcers that form slowly enlarge and become extensive lesions. After these fragments of burning metal are removed, the resultant wound must be treated as alkali burns.

Phosphorus Burns

White phosphorus is a waxy translucent substance that ignites spontaneously on contact with air. It is usually preserved under water and becomes a liquid at 44°C. White phosphorus is used in the construction of military weapons and fireworks and is a component in insecticides and rodent poisons.[33] White phosphorus is the most common form of phosphorus found in chemical burn (Fig. 22.9).

Phosphorous pentachloride is a yellowish white fuming crystalline mass with a pungent odor. It is widely used as a catalyst, a chlorinating and a dehydrating agent. There is only one report of phosphorus pentachloride chemical burn in the literature, so this agent is not extensively discussed.

Following an explosion of white phosphorus munitions, flaming droplets of white phosphorus are flung about widely and dense clouds of white smoke with a typical "garlic-like" smell are produced. The flaming pieces of phosphorus cause primarily thermal tissue damage but may also cause high-speed projectile injuries. The smoke is made by the oxidation of phosphorus to phosphoric acid, which can damage the lungs as well as the skin. Some "tracer" bullets have white phosphorus as a major component. This may lead to absorption of retained phosphorus fragments after either an explosion of a phosphorus-based shell or after a bullet wound with a tracer bullet.

White phosphorus is highly fat soluble and is easily absorbed from phosphorus particles driven into the subcutaneous lipid tissues or from the gastrointestinal tract if ingested. The two most common systemic effects are hepatotoxicity and renal damage, but changes in blood phosphorus and calcium levels are also noted. ECG changes with prolongation of the QT interval, ST segment depression, T-wave changes and bradycardia are also noted. Sudden death[34] has been reported in patients with reversed calcium-phosphorus ratios but is fortunately rare. Monitoring for electrocardiographic abnormalities and changes in serum calcium and phosphorus is appropriate for patients with significant wounds. In cases where systemic absorption is suspected, BUN, creatinine, serum phosphorus and liver enzymes should be assessed frequently.

Flaming white phosphorus is extinguished by immersing the pieces in water. Surface particles and particles embedded in clothing should be promptly removed. During transport, the emergency medical team should cover the burned areas with moistened cloths to prevent further burning. This is the same method that the armies of Israel and the United States recommend for transport of the patient burned by white phosphorus. Irrigation may help to remove surface particles, but is not effective against those particles driven deep into tissue by blast effects.

The wound may then be washed with one percent copper sulfate solution. The copper sulfate solution combines with phosphorus to form copper phosphate, which can be readily identified as black particles. These black pieces can be easily debrided. If the particles are not debrided, they are absorbed and systemic effects ensue. This is a controversial step and some investigators feel that it is not required and may even be detrimental to the patient. Certainly, once debridement is completed, no further copper sulfate solution is needed.

The copper sulfate solution should not be used for a prolonged period, because of the risk of systemic copper poisoning. Systemic copper poisoning can be manifested by vomiting, diarrhea, oliguria, intravascular hemolysis, hematuria, hepatic necrosis and cardiovascular collapse. The hemolysis is thought to be due to copper's inhibition of the erythrocyte hexose monophosphate shunt. This results in a hemolysis similar to that found in G6PD deficiency disease.

To minimize copper absorption, the wound may be irrigated with a solution of 5% sodium bicarbonate and 3% copper sulfate suspended in 1% hydroxyethyl cellulose. Wet dressings of copper sulfate in any form should never be applied to the wound. Following debridement, the wound is irrigated with copious amounts of water to remove the copper salts. Unfortunately, in the animal studies performed by Eldad and Simon, mortality was not decreased by use of this gel.

Other investigators irrigate the wound with sodium bicarbonate in order to neutralize the effects of the phosphoric acids. This also had no appreciable decrease in mortality in the experimental model.

Metabolic derangements have been noted with white phosphorus injuries. Hypocalcemia, hyperphosphatemia and ECG abnormalities are all noted. The electrocardiographic abnormalities include prolongation of the QT interval, bradycardia, and nonspecific ST-T wave changes.

Sulfur Mustard Gas

Sulfur mustard is a vesicant gas used primarily for warfare. It is a close relative of the cytotoxic nitrogen mustard compounds used for cancer chemotherapy. Sulfur mustard goes by several names: H, HD in the United States Military classification system, mustard, mustard gas or even Yperite (for the battle of Ypres where the first formal chemical warfare took place in 1915).

The toxic reactions of mustard occur when it forms highly reactive and unstable "onium" compounds that further cause inhibition of cellular glycolysis, alkylation of DNA purine bases (guanine and adenine) and breakdown of DNA. These effects occur within an hour of exposure to 19-33 mg/m^3 concentrations.[35]

Phenol Burns

Phenol is an aromatic acid alcohol that is a highly reactive and corrosive contact poison. The phenols have been used as bactericidal agents and are used in industry as bases for plastics and organic polymers. In the form of creosote, it is also used as a preservative for wood.

Phenols were first used as antiseptics by Lister in 1867 and their effects on tissues have been well studied. In either the vapor or the liquid form, phenol is rapidly absorbed through the skin surfaces. Phenol vapors are easily absorbed by the lungs. If the skin absorption is significant, the exposure may result in toxic effects on the cardiovascular and central nervous systems. These include:

- Central nervous system depression
- Hypotension
- Intravascular hemolysis
- Pulmonary edema
- Shock
- Death

The most common manifestation of systemic phenol poisoning is a profound fulminant central nervous system depression with coma, hypothermia, loss of vasomotor tone and respiratory arrest. In many cases, loss of consciousness may be noted within 30 minutes of skin contact. Cardiopulmonary collapse frequently accompanies this CNS presentation and is due to the cardiac depressant effects of phenol. In animal studies, as little as 0.625 mg/kg of phenol has caused death.

Phenol skin burns are characterized by initial pain, followed by numbness as the nerve endings are anesthetized. The skin then blanches and an eschar forms over the burned area during a period of hours. Phenols in strong concentrations rapidly produce a whitish slough, which turns to a greenish-black or copper-colored eschar. Coagulation necrosis of the dermis delays the absorption, but this delay is only temporary.

Treatment must be initiated immediately. Before washing the contaminated area, remove any liquid on the skin. Swabs, gauze pads or even clothing is acceptable for this. If polyethylene glycol is not available, glycerol may aid in the removal. Removal of the liquid should not wait until these materials are available.

Water irrigation is not recommended by some authorities. In severe phenol burns, the necrotic tissues may act as a temporary barrier that does not allow penetration of the water into the deeper layers of the injury. The water may also dilute the phenol and allow more rapid penetration into the skin.

Although water irrigation is effective for phenol burn, wiping the skin with undiluted polyethylene glycol 300 or 400 dissolves and removes the phenols more quickly[36] than water alone. Alcohols and glycerol are also effective in removal of the phenols. Polyethylene glycol solution is not irritating to the tissues and may safely be used to irrigate facial burns and eye injuries.

Alcohol Burns

Isopropyl alcohol is a commonly used disinfectant in hospital settings and is used for cleaning in home and industry. In infants and small children, it can be associated with skin burns. The mechanism of action is thought to be desertification of the skin. The degree of burn is related to the concentration of the alcohol, the duration of the exposure and the condition of the skin to which the alcohol has been applied. Premature infants are at most risk. Burns occur most often in areas of pressure and where the alcohol can pool, such as around the diapers.

Coal Tar Burns (Figs 22.15A to 22.16B)

Coal tar or bitumen is used for making roads and roofs. They are composed of different aliphatic, aromatic and heterocyclic compounds. They are used in molten form at very high temperatures (292°C). Whenever, the skin comes in contact with bitumen it rapidly cools down and adheres tightly to the skin. The heat contained within this layer of tar can continue to damage the underlying skin and cause deeper burns if not acted upon very quickly. However, if an attempt is made to peel off the bitumen it can cause further damage to the epidermis to which it bonds tightly. In case of small areas the tar is best left as such because the skin under it is usually sterilized by the heat and the layer of tar acts as a 'dressing' under which the skin heals spontaneously and the tar separates slowly in 2-3 weeks.

It is advisable to cool the skin with water for 30 minutes as a first aid measure to limit the damage to the skin. For extensive areas or if patient's face or limbs are involved circumferentially, the tar can be safely removed by wiping with swabs soaked in cooking oil or fat or butter or paraffin oil. It can be a time consuming and messy procedure and may not remove the entire material. The remaining bitumen

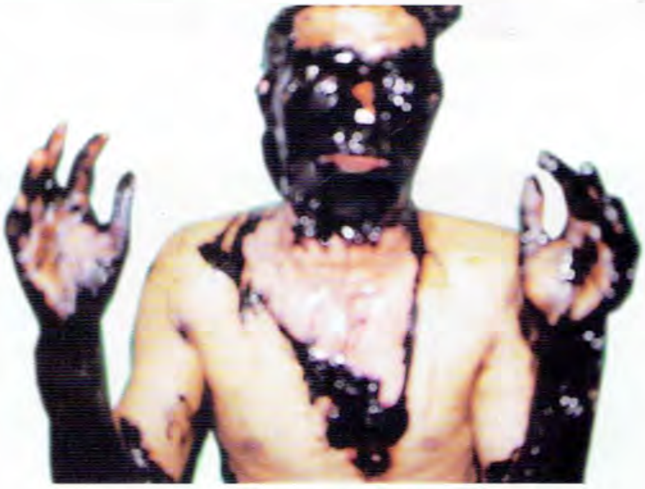

Fig. 22.15A: Coal tar burns involving face, chest and both arms

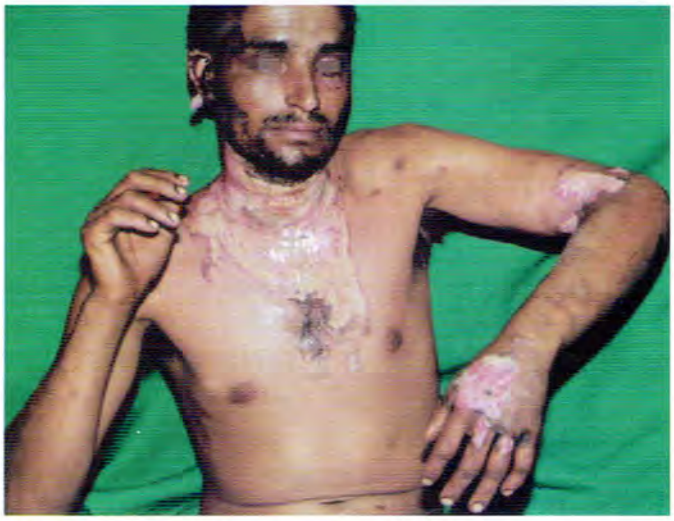

Fig. 22.15B: Same patient with completely healed face, arms and chest in 10 days indicating superficial burns

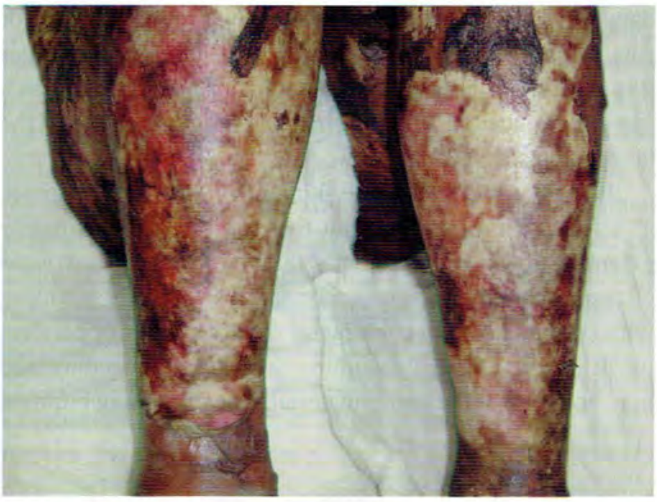

Fig. 22.15C: Same patient's legs with deep burns due to coal tar

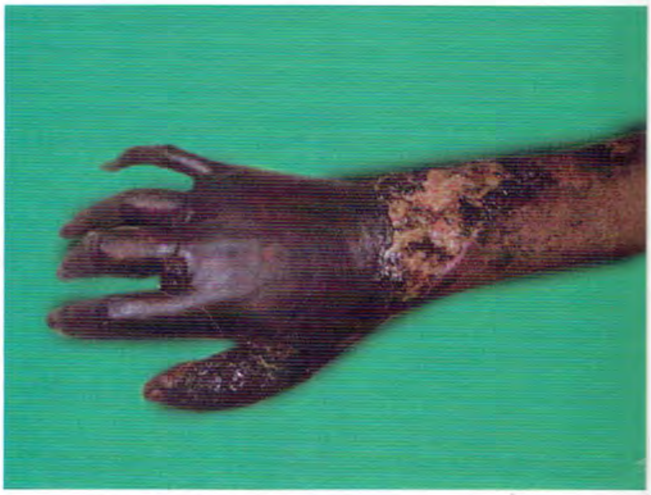

Fig. 22.16A: Patient with deep burns of hands due to coal tar

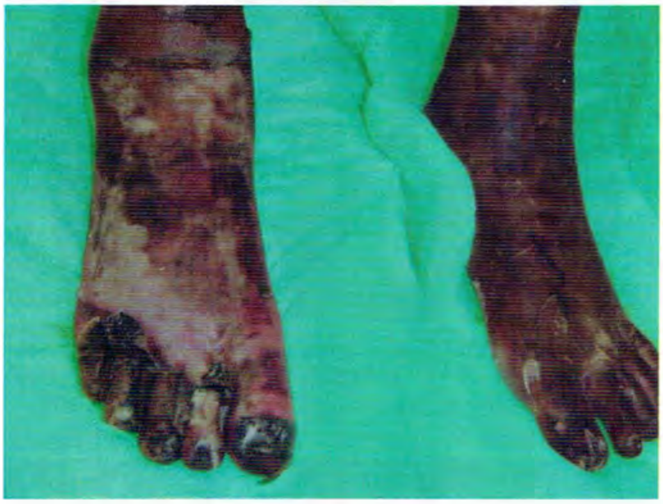

Fig. 22.15D: Same patient's feet with deep burns

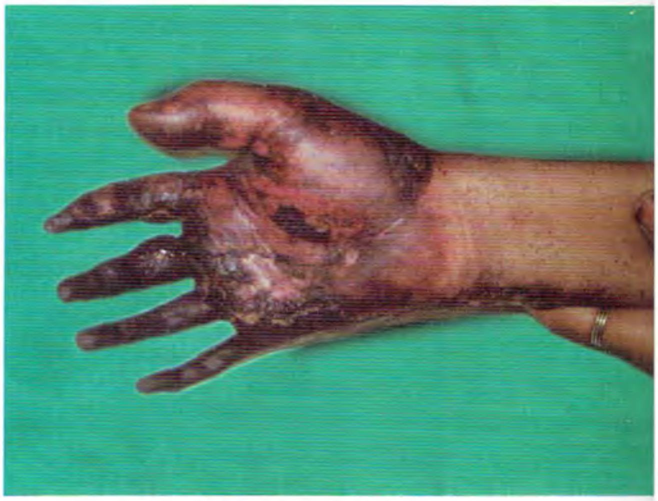

Fig. 22.16B: Same hand palmar aspect

can be trimmed away as it separates spontaneously over 3 weeks as the skin heals.

Many organic solvents like carbon tetrachloride, kerosene and petrol can readily dissolve bitumen but there is always a danger of the solvent causing chemical damage to the skin or being absorbed through the skin and poisoning the patient.

Molten Plastic Burns

Burns with molten plastic are seen in the plastic industry and like coal tar tend to adhere to the skin and may produce deep burns. It has to be removed surgically only.

Hot Metal Burns (Figs 22.17A to 22.18)

Molten metal burns are seen in furnace men, moulders and casters in the iron, steel, brass or aluminium industries. Since iron and steel have very high melting points (approaching 1500°C), the molten iron and steel usually produce full thickness burns. Aluminium has a lower melting point comparatively (650 °C); it produces deep dermal burns. Sometimes, if the temperatures are very high as it falls directly from the furnace on to a body part like dorsum of foot, the molten metal may even penetrate through and through like a bullet wound.

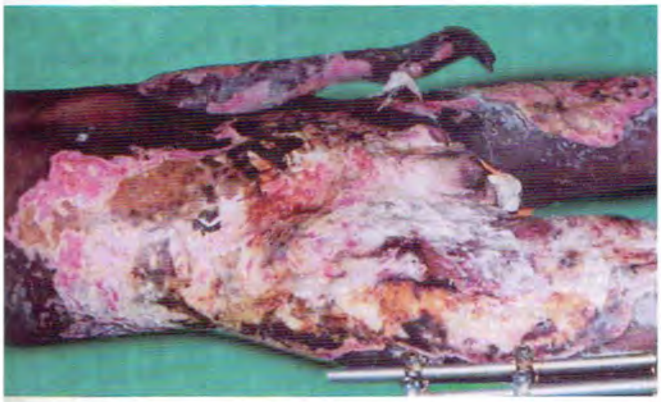

Fig. 22.17A: Deep burns of lower half of body and arms with crush injury of thigh due to fall of a hot metal box

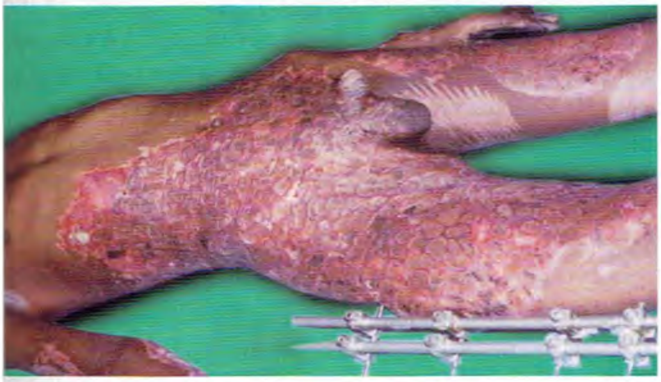

Fig. 22.17B: Same patient with completely healed wounds after grafting

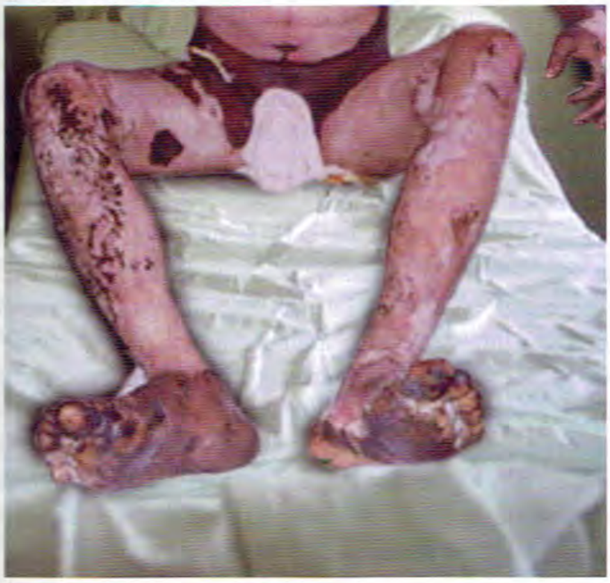

Fig. 22.18: Deep burns due to hot coal dust

Majority of these burns are due to accidental stumbling of the worker into a molten metal container or due to an explosion in the furnace. The main reason for explosion is water getting into the molten metal accidentally following which the molten metal splatters on to the workers' body parts or clothes which catch fire. The commonest body part involved are the feet as it may trickle down from the trousers inside the boots.

Molten metals typically produce full thickness burns which demand primary full thickness excision and grafting for best results. Molten aluminium burns may be deep dermal and for these cases tangential excision and grafting suffices. Some of the burns in which bones get exposed, may require flap cover.

SURGICAL TREATMENT IN CHEMICAL AND INDUSTRIAL BURNS

Small deep burns sustained from hot metal or slag is very common especially in steel industry. Though they are usually too small to cause serious threat to life or limb, they cause loss of huge man hours.[37] These small deep burns are best treated by excision of slough and skin grafting at the earliest under local anesthesia.

For moderate burns, early excision and skin grafting is the treatment of choice. However, it is not always feasible in Indian scenario because of constraints like non-availability of blood, shortage of operating time, etc. Patient should be taken for surgery after 48 to 72 hours after the patient is well resuscitated and adequate blood is arranged. Early excision and grafting is reserved only for those cases, which are full thickness burns. If the burns are of mixed nature or if there is any doubt regarding the depth it is better to wait for two weeks as by that time partial thickness burns start healing (Fig. 22.19). This reduces the area requiring grafting and thereby the blood transfusions required are also reduced.

In extensive burns also because of paucity of donor area, delayed excision and grafting (in third week post burn) is the treatment of choice, as by this time second degree superficial and some third degree deep burns heal. Healed second degree superficial areas can be utilized for harvesting skin graft in addition to the small available donor area in extensive burns. In metal burns with crush injuries also it is better to delay the grafting until third post burn week as the poly trauma is to be taken care of first (Figs 22.17A and B).

Cadaver skin and porcine skin is not available in India and cultured skin is still in its budding stage. So the preferred method is delayed skin grafting instead of early excision

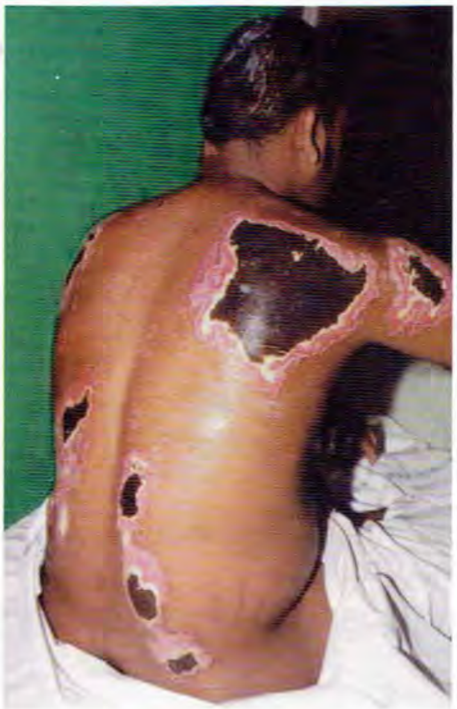

Fig. 22.19: Deep acid burns of back showing separation of eschar in the middle and healing of superficial burn from the periphery

and grafting in extensive burns. We like to take the patient for grafting in third week post burn. Usually by this time the partial thickness burns have either healed or they are in healing phase.

We prefer to give antibiotics as per the wound culture sensitivity reports to take care of both gram-positive and gram-negative bacteria. Antibiotics are started a day prior to surgery and continued till fifth postoperative day when primary dressing is done. Subsequently injectable antibiotics are stopped and switched over to oral antibiotics for another 3-5 days, if required. After the first dressing, subsequent dressings are done every alternate day or as per requirement till the wounds heal. Accordingly patient is asked to take bath around 11th to 14th postoperative day.

Subsequently patient is asked to do massage of the grafted and donor areas with a corticosteroid ointment twice a day. Physiotherapy, which is a continuous ongoing process, is continued during recovery phase. After massage and physiotherapy, patient is asked to use either pressure bandage or pressure garments. After two to three weeks, corticosteroid ointment is replaced with bland oils or creams. The duration varies in individual cases (4-6 months). Plain coconut oil massage is continued till scar matures (1-2 years). If there is extensive hypertrophy it is advisable to use silicon gel sheet lined pressure garments. For smaller hypertrophic areas intralesional triamcinolone injections are very effective at 4 weeks interval.

Surgical Procedure

Usually thin split thickness grafts are preferred. Donor areas are simply dressed using Vaseline gauze, dry gauze, cotton and pressure bandage in that order. Readymade available collagen sheets are used for donor areas, which are in near vicinity of burn wounds (In patients where separate donor areas like leg, thigh or back are not available). Donor dressings are not changed. They are opened after 11 to 14 days, when patient is asked to take bath. In extensive burns when grafting is to be done in multiple sittings same donor area can be used for graft harvesting after 3 weeks. In the authors unit burn wound is injected with saline adrenaline and gentamicin for proper hemostasis in the beginning at least 10 minutes prior to excision. (1 ampoule adrenaline and 80 mg. of gentamicin is added to 500 ml of saline). 5 ml of this solution is injected every 5 cm square area. If patient is sensitive to adrenaline or anesthetist has some objections adrenaline 1 ampoule is added to 1000 ml of saline. While one surgeon harvests skin graft another one prepares the burn wound. The operating nurse prepares the graft for application by meshing it in a ratio of 1:2. We do not spread the meshed graft to its fullest unless the available graft is very less. The burn wound is excised tangentially using a Watson's modification of Humby's skin grafting knife or a silver knife (for small areas) till healthy bleeding surface is reached. After excision of slough, the bleeding surface is covered with wet saline soaked gauze pieces and pressure is applied. Any big bleeder is cauterized. If bleeding is not controlled by these methods we sometimes use hemostatic solution soaked gauze pieces on the bleeding surface and wait for 5 minutes. Meshed graft is then applied over these raw surfaces. Pressure is again applied with wet gauze to remove any clots beneath the graft. The wound is covered with a very thin layer of Vaseline gauze. Over this 4-6 layers thick Povidone iodine solution soaked gauze is applied. These gauzes are squeezed nicely before application and they help to soak any discharge from the wound. Over this, dry gauze and then a very thin layer of cotton is applied. Pressure bandage is then applied and Plaster of Paris splints are used for immobilizing the grafted area. Subsequent dressing changes are done as mentioned earlier.

In electrical burns immediate fasciotomy is done over limbs or trunk. Subsequently, debridement and wound coverage is done according to the situation. There are two schools of thoughts for dealing with high tension electric current injury of limbs (Entry wound). Some surgeons immediately debride and cover the wound with flap. Others wait for line of demarcation before undertaking surgery. Many studies have shown that early debridement and flap cover only increases the morbidity. Majority of patients end up with amputation(s) of the limb. In our hospital we wait for one or two weeks before taking the wound for final closure. If the hand or foot is totally gangrenous at the time of admission or if patent shows signs of uremia with hemoglobinuria and myoglobinuria without any signs of improvement with resuscitation, the amputation is done within 2-3 days with stump coverage immediately, if feasible, or later on. Every case should be weighed individually and dealt with accordingly. All exposed vital areas must be covered with locally available flaps or free flaps. Surgical treatment of chemical burns remains the same as of flame or scald burn.

SUMMARY

Burns is the severest form of injury the body sustains. It is more serious than road traffic accidents. However, its seriousness is often overlooked, as the patient remains conscious till the end. Burns is one of the causes of systemic inflammatory response syndrome (other causes being polytrauma, septicemia, etc.) involving most of the organs because of the various inflammatory mediators released. If septicemia occurs in burns patients, it becomes almost impossible to save the patient. All efforts must be directed to achieve three goals simultaneously, that is, prevention of infection, maintenance of nutrition and rehabilitation. All these three objectives go hand in hand; laxity in any one part may lead to loss of patient. The metabolism in burn patients may be as high as 150% of the normal individual. So the nutritional requirements of a burn patient are tremendously high to maintain the immunocompetence. Also, there is tremendous loss of protein from the denuded body surface so that the extra requirement should be added to the daily requirement (We use Curreri formula to calculate the calorie requirement in burn patient (25 kcal/kg + 40 kcal/% burn).

The calorie nitrogen ratio is kept as 120:1 to 100:1

Protein administration is usually 1 gm/kg + 3 gm % BSA burnt.

Enteral route is preferred over parenteral unless the patient is not cooperative or burn is more than 40% where parenteral is added to enteral to supplement extra requirement, or where gut is not functioning properly. The maximum permissible dose of antibiotic and the maximum permissible dosage schedule is to be started in burn patient because of the high metabolism. The exact bacteria and their spectrum must be known before starting the antibiotic and is better to use target specific antibiotics in combination to cover both gram-positive as well as gram-negative bacteria.

Physiotherapy is another very important component of therapy. It should start from 2nd day, continued throughout treatment and carried over to post healing phase to take care of respiratory problems, infective problems, prevention of various contractures and prevention of bed sores development, etc.

Summary of Treatment Measures for Specific Chemical Burns

Irrigation with Water

Chromic acid, cantharides, lyes and alkalies, hydroxides of potassium, sodium, barium, calcium etc, sulfosalicylic acid, acetic acid, cresylic acid, potassium permanganate, demethyl sulfoxide (DMSO), sodium hypochlorites, phenol, hydrofluoric acid, dichromate salts, tungstic acid, picric acid, trichloroacetic acid, formic acid and gasoline.

Calcium Salt Irrigation or Injection

Hydrofluoric acid.

Cover Burn with Oil

Sodium metal, lithium metal, potassium metal and mustard gas.

Cover with Water

Phosphorous metal.

Special Measures for Certain Chemicals

Sodium, potassium and lithium metals: Pieces must be excised.

Phenol: Polyethylene glycol wipes

White phosphorus: Copper sulfate irrigation

Alkyl mercury agent: Debride and remove vesicle fluid.

For Electrical burns: More IV fluids per percent burn, keep urine alkaline to dissolve the pigments, if required give mannitol. Immediate fasciotomy to release the compartment tightness, flap cover for coverage of vital structures like vessels, tendons, bones, etc. Amputation of limbs if damage is too extensive because better prosthesis are more useful than a non-functional natural limb.

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Chapter 23

Radiation Burns

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INTRODUCTION

Radioactivity was discovered by a French physicist Henri Becquerel in 1896 and soon it was observed that biological damage can be produced by absorption of energy from this radiation in living organisms.

Though the word 'Radiation' encompasses the entire spectrum of emissions from gamma rays and X-rays (ionizing radiations) to the ultra violet, visible and infra red light, microwave and Radio waves (non-ionizing radiations), only in this chapter it is used to describe the effects of ionizing radiation only.

World has witnessed one of the worst effects of radiation following the bombing of Hiroshima and Nagasaki in 1945. Thereafter, more than 400 accidents have taken place in different settings ranging from a nuclear reactor accident and tele-therapy equipments to stolen radioactive sources with resultant deaths and radiation burns in several people. With increasing use of radiation in diverse fields like agriculture, medicine, industry and power generation, the chances of coming across radiation burns, although remote, is not impossible. It is, therefore prudent to gain an insight into the management of injuries arising out of radiation exposures.

International Basic Safety Standards defines an accident as "any unintended event, including operating errors, equipment failures or other mishaps, the consequences or potential consequences of which are not negligible from the point of view of protection or safety" and a radiological accident as "an unforeseen event involving overexposure or contamination of person(s) and/or the environment by radioactive material".

The accidents at Chernobyl, Ukraine (April 1986), Goiânia, Brazil (September 1987), San Salvador, El Salvador (1989) and several others provided important information for the diagnosis, monitoring and treatment of radiation injuries.

TYPES OF ACCIDENT

An accident is called a nuclear accident when it involves nuclear fission especially in a nuclear reactor. A radiological accident, on the other hand, involves a release of ionizing radiation (X-ray equipment or sealed sources like Cobalt 60 (60 Co), Cesium137 (137 Cs) or Iridium 192(192 Ir) in tele-therapy equipments, industrial radiography cameras or irradiators) or discharge of radioactive materials (unsealed sources used in nuclear medicine and scientific research) into the environment.

Accidents may occur in the following situations:

a. A radioactive source (in tele-therapy equipments or industrial radiography cameras) may be stolen, lost or misplaced and may come into the possession of people thereby exposing them and possibly others to radiation.
b. A radioactive source (in tele-therapy equipments, industrial radiography cameras or irradiators) may fail to get retracted after use and may remain unshielded, exposing the operator to high levels of radiation.
c. A vial containing radioactive material may leak and the material may get dispersed exposing and often resulting in external and internal contamination of the person.

BASICS OF RADIATION

Radioactive materials emit energy in the form of alpha (α) particles, beta (β) particles and gamma (γ) rays. These radiations may interact with matter and give rise to the emission of X-rays and neutron particles. Certain machines operated at high voltages can generate X-rays and neutrons. The shorter wavelength of the neutrons or X-rays gives them a higher energy and ability to penetrate matter.

β particles are emitted by radionuclides such as hydrogen-3 (3H) and phosphorus-32 (32P) and neutron particles in general travel more slowly than beta particles, but being heavier particles they consequently have higher energies.

As radiation travels through human tissue, it collides and interacts with the component atoms and molecules resulting in the atom or molecule to be altered and becoming an ion. The radiation leaves a trail of ionized atoms and molecules in its course.

Alpha (α) particles are heavy particles (nuclei of Helium atom) and carry a positive charge. They can travel within a few cm in air or about 50 micron (millionth of a meter, μm) in human soft tissue and can be easily stopped by a thin sheet of paper. Thus, they do not even penetrate the outer horny layer of skin and therefore do not cause any external radiation damage. However, they can cause great damage if they get incorporated in the body following inhalation, ingestion or through open wounds due to the intense ionization they give rise to.

Beta (β) particles are light particles (electrons) and can travel several meters in air and about a cm in tissues and can be stopped by a thin sheet of aluminium or 1 cm of plastic. As they deposit their energy in a smaller thickness of tissue, they can cause greater damage to the superficial layers of the skin giving rise to burns (also called burns).

Gamma (γ) and X-rays have a range of many meters in air and many cm in tissues and eventually pass through a human body and can be attenuated by about 20 cm of concrete. As they penetrate deeper into the body, they cause harm to the deeper structures and vital organs, including the bone marrow, giving rise to systemic effects of "Acute radiation syndrome (ARS)" apart from causing skin damage.

Neutrons interact with atoms and molecules in complex ways causing ionization indirectly through the charged particles (α, β, γ) they release upon interaction. They have a range of many meters in air and many cm in the tissues and eventually get absorbed or pass through a human body (Table 23.1).

RADIATION BIOLOGY

Ionizing radiation can have a direct action on the DNA within the cell by breaking the bonds between the atoms. Indirectly, ionization of water molecules produces free radicals which are capable of attacking the coded genetic information in the cell nucleus, disrupting the cell's chemistry and function or physically rupturing membranes containing the digestive enzymes. Natural repair mechanisms can limit the damage and improve the cell's chance of survival. However, if the repair is incomplete, the cell may become incapable of dividing and may eventually die or may behave abnormally leading to development of cancers.

The sensitivities of different tissues of the body is different, being the highest in lymphocytes, the fast dividing cells of the bone marrow and the cells lining the intestinal villi. The nerve tissue, by contrast, is the most radio resistant tissue.

RADIATION UNITS

The unit of radiation dose is defined as energy absorbed per unit mass of matter with which radiation reacts (measured in Joules/kg). The SI unit of the absorbed dose is Gray (Gy), the earlier unit for which was rad (1 Gy = 100 rad).

The biological damage (cell killing, transformation, chromosomal damage) caused by different radiations may vary and hence a quantity called "Relative Biological Effectiveness (RBE)" was introduced. The ICRP (International commission for radiation protection) has recommended a quantity called "Quality factor (Q)" or "Radiation weighting factor ($W_{R)}$" to find the dose-equivalent (Table 23.2).

Table 23.1: Characteristics of different types of radiation

Type of Radiation	*Relative mass in amu(atomic mass unit) and charge*	*Range in air*	*Absorber material which can be used as a shield*
Alpha	4 amu; (+2)	Few cm.	Paper
Beta	Negligible; (-1)	Few meters	Plastic or Aluminum
Gamma	Zero; (0)	Very Large	Lead, water or concrete
Neutrons	1 amu; (0)	Very Large	Cadmium or Boron for Thermal Neutrons; Paraffin followed by Cadmium or Boron for Fast Neutrons

Table 23.2: Radiation weighting factor for different radiations

Type of radiation	*Radiation weighting factor: W_R*
Gamma	1
Beta	1
X-rays	1
Alpha	20
Heavy Ions	20
Neutrons	5 to 20 depending on the energy of the neutrons.
Protons	5

The dose-equivalent is derived by multiplying the dose with the Radiation weighting factor. The SI unit for dose-equivalent is Sievert (Sv), the earlier unit for which was rem (1 Sv = 100 rem).

RADIATION INJURIES

Radiation injuries may manifest as a localized or whole body exposure or as external deposition or incorporation (internal contamination) of radioactive material in the body. The effects may be dependent on the dose and type of radiation received and the area of the body involved. Careful history and early clinical findings may guide the treating physician.

ACUTE RADIATION SYNDROME

Whole-body Exposure

A single acute high dose (≥ 1 Gy) exposure of penetrating radiation like radiation or X-rays to the major portion of the body can give rise to a clinical entity of "Acute Radiation Syndrome (ARS)". The condition is heralded by a prodromal phase which may be marked by nausea, vomiting (Hematopoietic involvement), diarrhea (Gastrointestinal involvement), prostration, hypotension (Cardiovascular involvement), ataxia and convulsions (Central nervous system involvement) in increasing order of severity. The time of onset and severity may give an indication of the system involved (Tables 23.3 to 23.5).

The prodromal period may be followed by a latent period of 2-3 weeks during which the person may feel better (at higher doses the latent period may be absent) only to be followed by a stage of manifest illness.

ARS may be classified according to the system most affected as:

Hematopoietic syndrome (1-10 Gy): The injury to bone marrow results in thrombocytopenia and granulocytopenia with resultant hemorrhages and infections needing blood component therapy and treatment of opportunistic infections. Hematopoietic Growth Factors—-G-CSF(Granulocyte colony stimulating factor), rhGMCSF (recombinant human granulocyte macrophage colony stimulating factor), rhIL6 (recombinant human interleukin -6) have been used to stimulate the residual bone marrow. Doses higher than 9 Gy may require a Bone Marrow Transplant (BMT) or Peripheral Blood Stem Cell Transplant (PBSCT).

Gastrointestinal syndrome (10-20 Gy): The injury to intestinal mucosal stem cells gives rise to death due to severe diarrhea, electrolyte disturbances, and sepsis.

Cardiovascular syndrome (> 30 Gy) and CNS syndrome (> 30-50 Gy): The hypotension and cerebral edema and injury to various other vital organs results and only terminal care remains possible.

Table 23.3: Dose-effect relation after acute whole-body radiation from gamma rays or X-rays

Whole-body absorbed dose	*Effect**
0.05 Gy	No symptoms
0.15 Gy	No symptoms, but possible chromosomal aberrations in cultured peripheral-blood lymphocytes
0.5 Gy	No symptoms (minor decreases in white-cell and platelet counts in a few persons)
1 Gy	Nausea and vomiting in approximately 10 percent of persons within 48 hour after exposure
2 Gy	Nausea and vomiting in approximately 50% of persons within 24 hour, with marked decreases in white-cell and platelet counts
4 Gy	Nausea and vomiting in 90% of persons within 12 hr, and diarrhea in 10% within 8 hour; 50% mortality in the absence of medical treatment
6 Gy	100% mortality within 30 days due to bone marrow failure in the absence of medical treatment.
10 Gy	Approximate dose that is survivable with the best medical therapy available
>10-30 Gy	Nausea and vomiting in all persons in less than 5 minutes; severe gastrointestinal damage; death likely in 2 to 3 weeks in the absence of treatment
>30 Gy	Cardiovascular collapse and central nervous system damage, with death in 24 to 72 hour

*Data are from Gusev et al.

Table 23.4: Skin changes after single acute localized exposure*

Absorbed Dose	*Change*
3-4 Gy	Epilation in 2–3 weeks
10-15 Gy	Threshold for erythema; appears 18-20 days after exposure at lower doses but may appear within a few hours at higher doses
20 Gy	Moist desquamation with possible ulceration
25 Gy	Ulceration with slow healing
30-50 Gy	Blistering and necrosis at 3 weeks
100 Gy	Blistering and necrosis at 1-2 weeks

*Data are from Gusev et al.

Table 23.5: Time of onset of clinical signs of skin injury-depending on the dose received

Stage/symptoms	*Dose range (Gy)*	*Time of onset (days)*
Erythema (Transient)	3	Hours to days
Erythema (Fixed)	6-10	14-21
Epilation	>3	14-18
Dry desquamation	8-12	25-30
Moist desquamation	15-20	20-28
Blister formation	15-25	15-25
Ulceration (within skin)	>20	14-21
Necrosis (deeper penetration)	>25	>21
Gangrene	>30	

Localized Exposure

Handling of highly radioactive sources (e.g. placing a hand in the beam of industrial radiography camera) may give rise to a very high dose locally. However, the torso being spared, the dose may not give rise to symptoms of whole body exposure or ARS and the systemic manifestations may be absent. The signs of radiation burn may take several days to appear. The vascular insufficiency caused by endarteritis obliterans may result in breaking down of previously healed skin.

INVESTIGATIONS

For Dose Estimation

History of exposure to radiation in the form of X-rays or gamma rays from teletherapy/industrial radiography source may be elicited. Absence of contact with corrosives, heat or insect bites should arouse suspicion of a radiation injury.

Physical Dosimetry

1. If the person has a personal thermo luminescent dosimeter (TLD), it should be collected and sent for reading to give an indication about the dose received by the person.
2. Accident reconstruction—A detailed history of the accident should be recorded. The exposure geometry which includes activity of the source, the distance from the source and the approximate time of exposure can give an idea of the dose received by the person.
3. The use of electron spin resonance method can be helpful to estimate the dose incurred. This can be used with samples like dental enamel, clothes, buttons, earrings or any organic substance exposed to radiation.

Biological Dosimetry

1. 6 hourly peripheral blood counts for absolute lymphocyte counts when plotted on a graph called 'Andrew's nomogram' give an accurate indication of the dose.
2. Chromosomal analysis of cultured lymphocytes
3. Semen analysis – Estimation of sperm counts immediately after exposure (as a base line reading) and 40-60 days thereafter.
4. Slit-lamp examination of eyes.

Investigative Modalities for Assessing the Severity of Cutaneous Radiation Injury

1. Serial color photographs– Even a detailed description is not enough to give a proper indication about the progress of the lesion as a function of time. While assessing the damage, serial color photographs (calibrated by using a piece of white paper) may be valuable.
2. Ultrasound (7.5-20 MHz B-scan sonography)– A 20 MHz probe is used for evaluating epidermis, dermis and subcutaneous fat tissue up to the depth of 10 mm whereas a 7.5 MHz probe can be used for evaluation of dermis, subcutaneous fat tissue, muscle fascia and musculature. It is also useful for assessing the depth of ulcers and the extent of radiation fibrosis.
3. Vascular scintigraphy is a radio-isotopic method in which 99mTc pertechnate is injected intravenously to record the vascular circulation.
4. CT scan for assessing damage to deeper structures.
5. MRI– This non-invasive investigation can reveal fluid in the tissues (as a result of inflammation, edema or necrosis). The extent of ulcer can be estimated as also the extent of necrotic tissue and this can influence the decision about conducting a conservative necrectomy or a more radical amputation.

6. Thermography– The heat emitted by the skin is directly dependent on the surface temperature of the skin, which in turn is dependent on the cutaneous vascularity. Significantly lower skin temperatures are seen in necrosis. The different techniques available are liquid crystal contact thermography, infrared and microwave telethermovision.
7. Capillary microscopy– The capillaries of the stratum papillare can be evaluated by using this non-invasive method, the capillaries of the nail fold of the fingers or toes being dilated in the manifest stage whereas they are few and smaller in the chronic phase of cutaneous syndrome.
8. Profilometry
9. Histology– In the manifest stage, dilated blood vessels, edema and neutrophilic and eosinophilic infiltration is observed whereas the chronic/late stage is set apart by epidermal atrophy/hypertrophy, dermal fibrosis, dilated blood and lymphatic vessels, pigmentary changes like hypo/hyperpigmentation and a deficit of hair follicles.

   Investigations required for effective treatment are:
   I. Peripheral blood counts
   II. Bacterial culture and antibiotic sensitivity tests

MEDICAL TREATMENT

As the cutaneous injury may be associated with ARS, the following care may be required

1. Supportive care –
   a. Antiemetics
      i. Antihistaminics (dimenhydrinate) – Poor efficacy.
      ii. Serotonin selective 5-HT$_3$ receptor antagonists (e.g., ondansetron, tropisetron, granisetron) – High effectiveness.
      iii. Dopamine-D2 receptor antagonists (e.g., metaclopramide) – Good efficacy.
   b. Analgesics
      i. Analgesics (paracetamol).
      ii. Non-steroidal anti-inflammatory drugs (ibuprofen, diclofenac, meloxicam). Aspirin is avoided as it can cause bleeding in thrombocytopenia.
      iii. Opiates (codeine, tramadol, pethidine, morphine, buprenorphine in increasing order of effectiveness). These may be combined with antiemetics to offset the emesis caused by opiates.
   c. Nutrition – Severe nausea may necessitate use of parenteral nutrition in the form of prefabricated solutions or total parenteral nutrition through a central venous line.
   d. Antibiotics, antifungals, antivirals – In case of associated ARS with attendant leucopenia and thrombocytopenia, infection of the respiratory tract, mucous membranes, skin ulcers and internal organs can take place. Mostly, these infections are a result of preexisting endogenous organisms. This has to be adequately countered by use of oral drug combinations like metronidazole and ciprofloxacin for sterilization of the gut. Antibiotics can be administered as per the results of the antibiotic sensitivity tests. Empirically, broad spectrum penicillins combined with third generation cephalosporins or carbapenems alone can be given. If there is an evidence of an infection not responding to antibiotics, antifungals like fluconazole should be considered. Antivirals like acyclovir or ganciclovir may have to be employed in herpes simplex or cytomegalovirus infections respectively.
2. Skin treatment
   i. Anti-inflammatory cream – Linoleic acid cream
   ii. Non-atrophogenic local steroid/ Systemic steroid (equivalent to prednisolone 0.5-1.0 mg/kg body weight)
   iii. Analgesics (as above)
   iv. Non-sedative mast-cell stabilizer – Loratadine
   v. Antibiotic creams / Systemic antibiotics
   vi. Heparin
   vii. Argon laser for ablation of telangiectasias
   viii. Lioxasol cream increases cell proliferative activity in the skin
   ix. Tretinoin cream
   x. Topical / Systemic superoxide dismutase (SOD)
   xi. Systemic pentoxiphylline
   xii. Systemic alpha-tocopherol
   xiii. Systemic proteinase inhibitors like Aprotininum (Gordox)
   xiv. Systemic interferon gamma
   xv. Topical tetrachlorodecaoxide (TCDO)
   xvi. Solcoseril gel has regenerative properties
   xvii. Actovegin is obtained by ultra-filtration of calf blood. It contains inositol phospho-oligosaccharides (IPOs), which are known to increase uptake and utilization of oxygen by hypoxic tissues and cells by promoting mitochondrial respiratory function, decreasing the formation of lactate, and in turn, protecting hypoxic tissue.

xviii. Hydrocolloid dressings
xix. Topical thrombocytic growth factors
xx. Semi-synthetic skin equivalent - Integra

The various degrees of radiation burns described are as follows:

Erythema and dry desquamation: Treatment is purely symptomatic. Severe erythema associated with edema is treated with lotions or sprays containing hydrocortisone to relieve the symptoms. Tight clothing should not be worn over the affected part.

Moist desquamation needs daily sterile protective dressings and bathing of the affected skin in antiseptic solutions. Antibiotic creams can also be used to prevent bacterial infections.

Ulceration requires isolation of the limb in a sterile environment. A daily dressing and bathing of the ulcer in antiseptic solutions is recommended. Analgesics or stronger opioids may be necessary. Topical or systemic antibiotic therapy should be considered if secondary infection is suspected or verified by bacterial culture.

Necrosis needs excision followed by skin grafts or other types of skin cover. Unless the underlying vasculature is intact, skin grafting may not be possible and a myocutaneous flap or pedicle flap may be required. Surgery in the form of ulcerectomy, necrectomy and amputation may be needed, the extent, timing and type of surgical intervention being determined on a case to case basis as the lesion progresses.

Indications for amputation depend on the following determinants:-

a. Intractable pain
b. Size and location of the injury
c. Degree of control of infection
d. Very severe destruction of underlying tissues with vascular damage
e. Value of the part

Not only does the latent period for development of signs and symptoms but also the intensity and duration of lesions depend on the dose and the type of radiation received. Thus, at a high dose (>100 Gy) the manifestations may develop early (within a week), whereas with a low dose they may take weeks to develop.

CUTANEOUS RADIATION SYNDROME (FIGS 23.1A TO 23.5B)

The basal cells of the epidermis including those cells situated within the canal of the hair follicle are the cells, which are damaged resulting in denudation of the epidermis resulting in manifestations of Cutaneous Radiation Injury syndrome (CRI). The erythema is a result of dilatation of superficial blood vessels due to an inflammatory reaction (Table 23.6).

Grading

Grade 1

Prodromal stage may start with itching and a brief transient erythema usually subsiding within 36 hours of exposure. The true erythema (a second wave of erythema also referred to as fixed erythema) appears around 5 days post-exposure and lasts a few weeks. Damage to sweat and sebaceous glands may give rise to a dry, scaly desquamation of the skin in 20-30 days, which resolves within a few weeks.

Treatment: Outpatient Care

Topical anti-inflammatory, anti-proliferative, non-atrophogenic glucocorticoids.
Systemic non-sedative anti-histamines.

Investigations: Ultrasound to determine changes in thickness of the skin and a basal thermography may show hyperthermic and hypothermic areas, which could serve as a reference for long-term follow-up.

Grade 2

Skin surface affected < 10% body surface. This phase consists of blisters which generally form around 4 weeks post exposure and can result in moist desquamation which can even occur without prior blisters due to depletion of epidermal stem cells. This may be associated with transient hair loss (around 2 weeks).

Treatment: Outpatient Care

Topical anti-inflammatory, anti-proliferative, non-atrophogenic glucocorticoids.
Systemic non-sedative anti-histamines
Linoleic acid creams
Puncture of blisters
Non-adherent dressings
Prevention of infections as per antibiotic sensitivity tests

Investigations: 20 MHz sonography to determine changes in thickness of the skin

Thermography to detect hyperthermic and hypothermic areas as a reference for regular follow-ups.

Table 23.6: Grades of cutaneous radiation injury (CRI)

Grade	*Skin dose**	*Prodromal stage*	*Latent stage*	*Manifest illness stage*	*Third wave of erythema†*	*Recovery*	*Late effects*
I	> 2 Gy (200 rads) ‡	1-2 days postexposure or not seen	no injury evident for 2–5 weeks post-exposure[§]	• 2–5 weeks postexposure, lasting 20-30 days: redness of skin, slight edema, possible increased pigmentation • 6-7 weeks postexposure, dry desquamation	Not seen	Complete healing expected 28-40 days after dry desquamation (3-6 months postexposure)	• Possible slight skin atrophy • Possible skin cancer decades after exposure
II	> 15 Gy (1,500 rads)	6-24 hours post-exposure with immediate sensation of heat lasting 1-2 days	No injury evident for 1-3 weeks post-exposure	• 1-3 weeks postexposure; redness of skin, sense of heat, edema, skin may turn brown • 5-6 weeks postexposure, edema of subcutaneous tissues and blisters with moist desquamation • Possible epithelialization later	• 10-16 weeks postexposure, injury of blood vessels, edema, and increasing pain • epilation may subside, but new ulcers and necrotic changes are possible	Healing depends on size of injury and the possibility of more cycles of erythema	• Possible skin atrophy or ulcer recurrence • Possible telangiectasia (up to 10 years postexposure) • Possible skin cancer decades after exposure
III	> 40 Gy (4,000 rads)	4-24 hours post-exposure, with immediate pain or tingling lasting 1-2 days	None or less than 2 weeks	• 1-2 weeks postexposure: redness of skin, blisters, sense of heat, slight edema, possible increased pigmentation • Followed by erosions and ulceration as well as severe pain	• 10-16 weeks post-exposure: injury of blood vessels, edema, new ulcers, and increasing pain • possible necrosis	Can involve ulcers that are extremely difficult to treat and that can require months to years to heal fully	• Possible skin atrophy, depigmentation, constant ulcer recurrence, or deformity • Possible occlusion of small vessels with subsequent disturbances in the blood supply, destruction of the lymphatic network, regional lymphostasis, and increasing fibrosis and sclerosis of the connective tissue

Contd.

Contd.

Grade	*Skin dose**	*Prodromal stage*	*Latent stage*	*Manifest illness stage*	*Third wave of erythema†*	*Recovery*	*Late effects*
							• Possible telangiectasia • Possible skin cancer decades after exposure
IV	> 550 Gy (55,000 rads)	Occurs minutes to hours postexposure, with immediate pain or tingling, accompanied by swelling	None	• 1-4 days postexposure accompanied by blisters • Early ischemia (tissue turns white, then dark blue or black with substantial pain) in most severe cases • Tissue becomes necrotic within 2 weeks following exposure, accompanied by substantial pain	Does not occur due to necrosis of skin in the affected area	Recovery possible following amputation of severely affected areas and possible skin grafts	• Continued plastic surgery may be required over several years • Possible skin cancer decades after exposure

*Absorbed dose to at least 10 cm^2 of the basal cell layer of the skin

†Especially with beta exposure

‡The Gray (Gy) is a unit of absorbed dose and reflects an amount of energy deposited in a mass of tissue (1 Gy = 100 rads).

§Skin of the face, chest, and neck have a shorter latent phase than the skin of the palms of the hands and the skin of the feet.

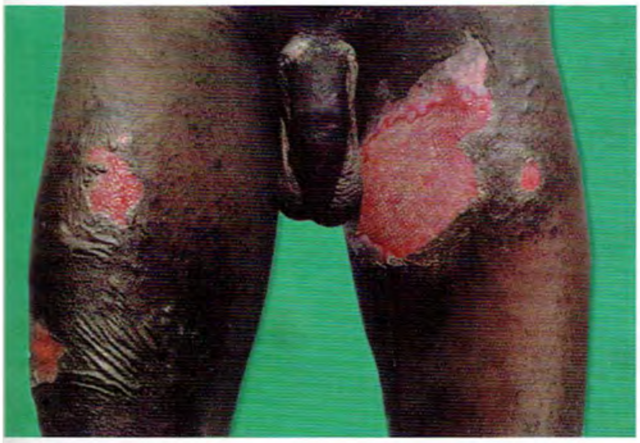

Fig. 23.1A: Evolution of cutaneous radiation syndrome following accidental exposure to an industrial radiographic device showing fixed erythema and blistering 4 weeks postexposure. Erythema is best appreciated in fair skinned individuals. The transient erythema cannot be appreciated in the dark skinned. In the dark skinned, fixed erythema appears darkly pigmented

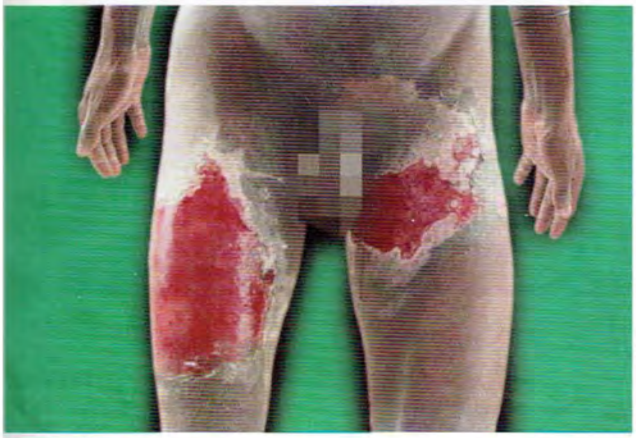

Fig. 23.1B: Ulceration 5 weeks postexposure

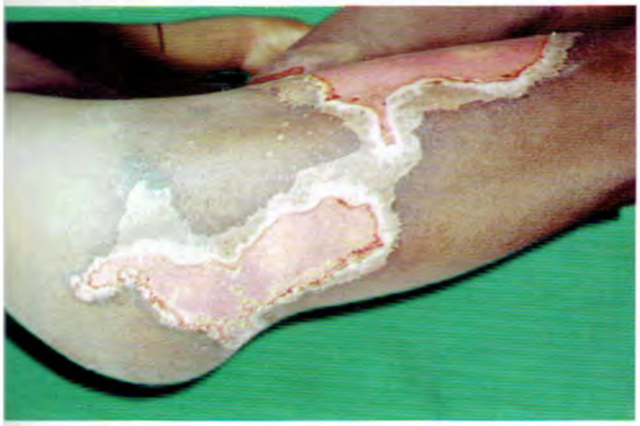

Fig. 23.1C: Ulceration and partial healing 6 weeks postexposure

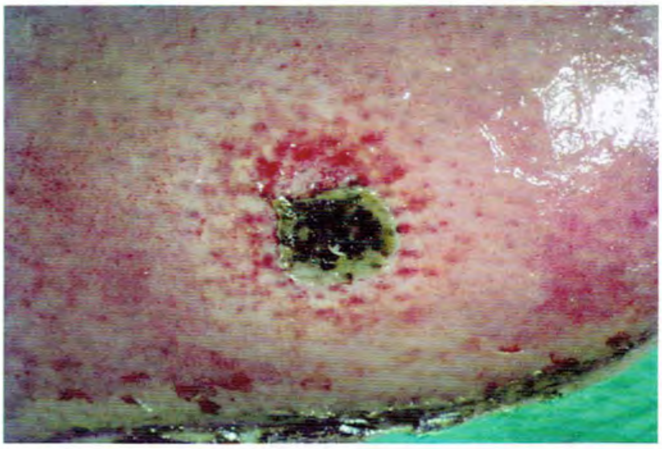

Fig. 23.1D: Ulceration with central necrosis 8 weeks postexposure

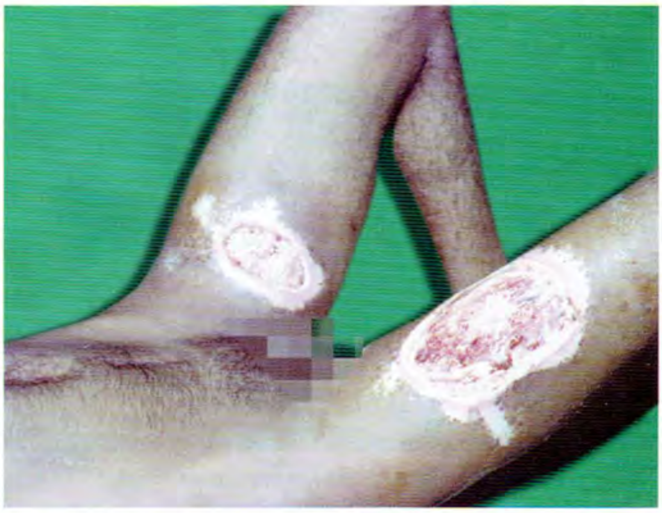

Fig. 23.1E: Same wound 14 weeks postexposure showing deepening of ulceration up to the fascia and muscle

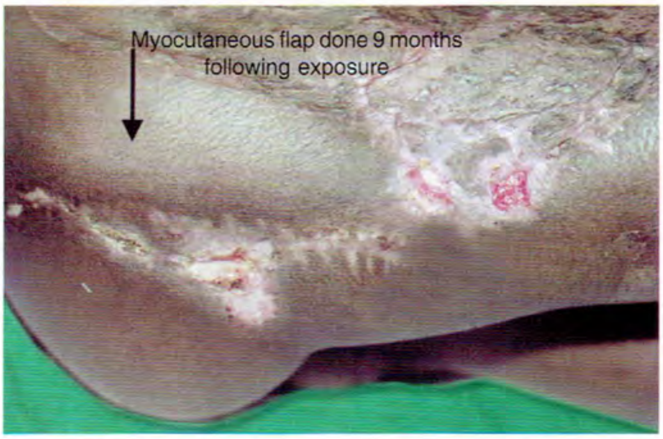

Fig. 23.1F: Even after 9 months with surgical cover few areas of ulceration still remain

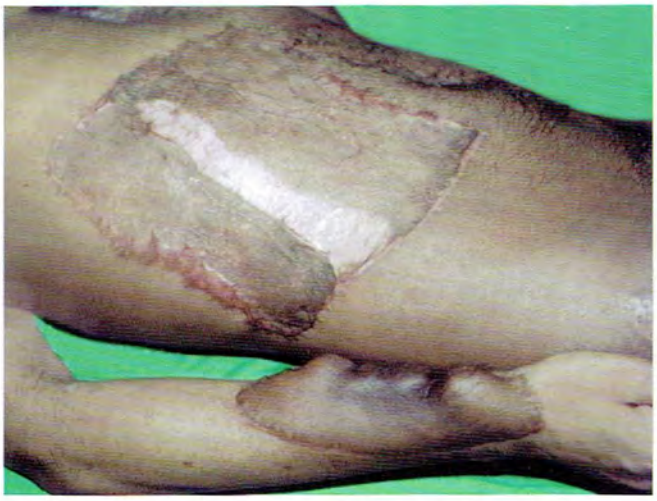

Fig. 23.1G: Same patient with abdominal flap cover following radiation burn wound over forearm

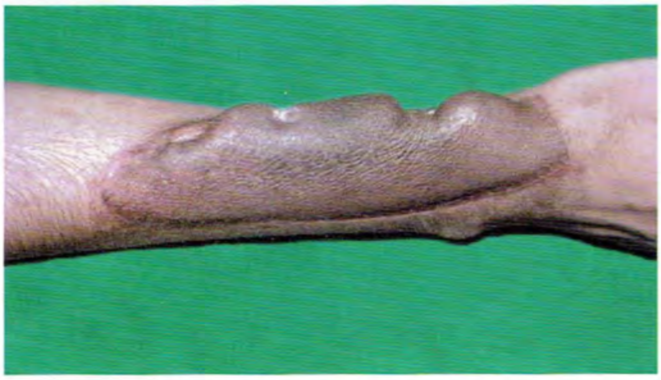

Fig. 23.1H: Close up view of the flap on forearm

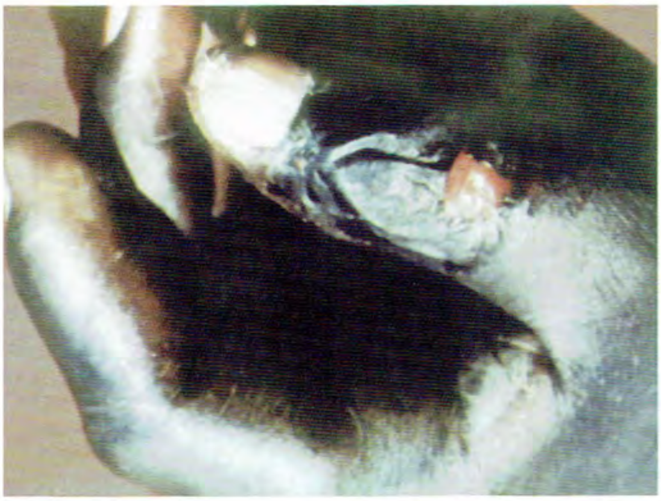

Fig. 23.2: Acute radiation burn of left hand following a 20 Gy exposure to a Co-60 source

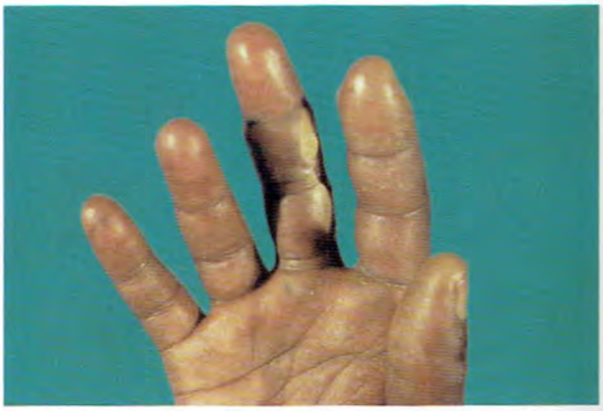

Fig. 23.3: Hand exposed to a radiographic unit showing changes in middle finger and thumb 6 weeks after exposure

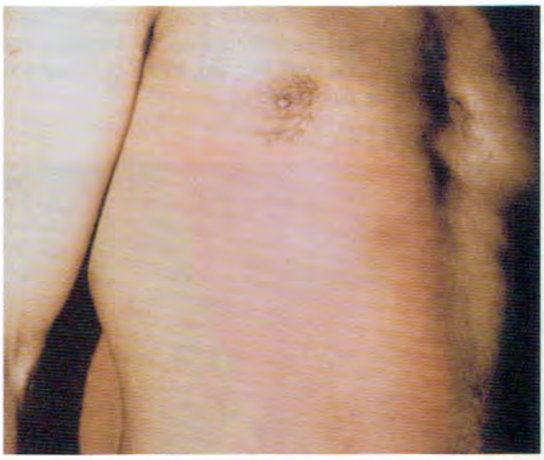

Fig. 23.4A: Iridium-192 source (185 GBq, 5 Ci) mounted in a pen-size source holder for industrial radiography was placed in the pocket of the worker's overall and kept there for about two hours (*Courtesy:* Figure 23.4A to C from How to recognize and initially respond to an accidental radiation injury (pamphlet), IAEA, Vienna, Austria, 2000) showing early erythema 5 days after the exposure

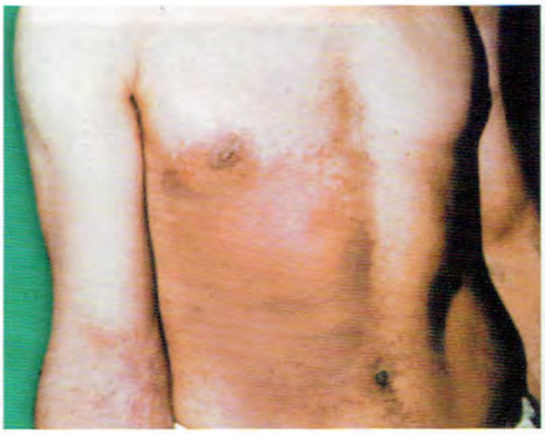

Fig. 23.4B: Erythema 11 days after exposure

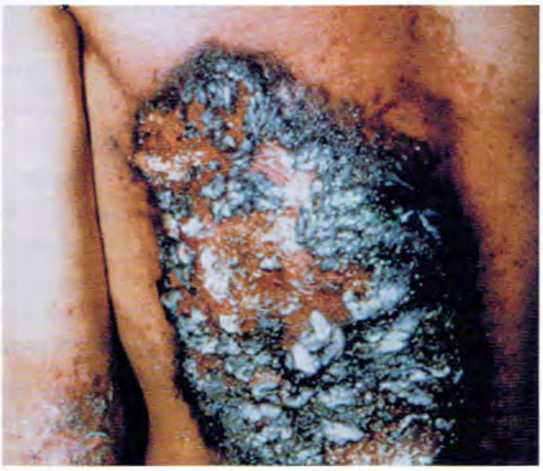

Fig. 23.4C: Desquamation and skin necrosis 21 days after exposure

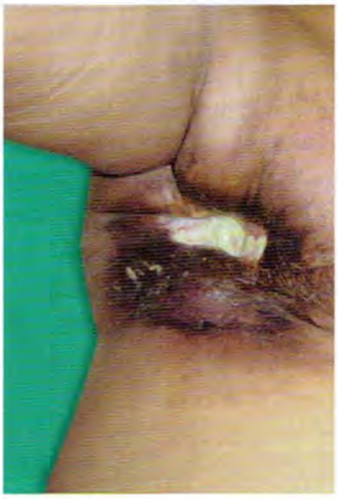

Fig. 23.5A: Non-healing ulcer on right chest following radiotherapy given postmastectomy (*Courtesy:* Editor)

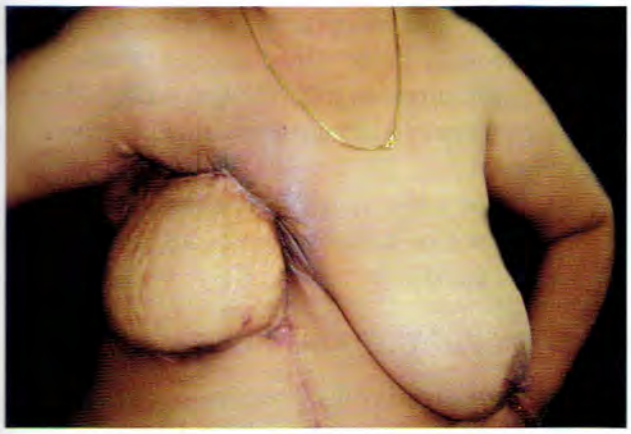

Fig 23.5B: TRAM flap done for cover of the same defect (*Courtesy:* Editor)

Grade 3

Skin surface affected 10-40% body surface. This phase consists of severe swelling, which is caused by increased vascular permeability and loss of fluids in the third space. Blisters form around 5 days postexposure resulting in ulcer or necrosis. Recovery may follow. However, it is incomplete and the sequelae may be alopecia, fibrosis, contractures, pigment changes, increased vulnerability to trauma and repeated breakdown of healed areas.

Treatment: Inpatient care

Topical antibacterial agents

Linoleic acid creams

Systemic anti-inflammatory, anti-proliferative, non-atrophogenic glucocorticoids to reduce edema.

Analgesia

Aspiration of fluid from blisters

Debridement of necrotic tissues

Non-adherent dressings

Prevention of infections as per antibiotic sensitivity tests

Investigations: 7.5 MHz sonography to determine changes in thickness of the skin

Doppler ultrasound to assess damage to underlying larger vessels

Sonography to assess the depth of the lesion

MRI to detect damage to underlying muscles

Surgery may be required in the form of full-thickness skin grafts/flaps.

Grade 4

Skin surface affected >40% body surface. This phase consists of severe damage to not only epidermis and dermis but also to subcutaneous tissues, underlying muscles and perhaps bones. This is associated with severe swelling, which is caused by increased vascular permeability and loss of fluids in the third space. Bullae form within a few days post-exposure and can result in acute necrosis. Recovery is unlikely and leaves deficits like alopecia, fibrosis, contractures, pigment changes and increased vulnerability to trauma, thermal and pressure changes, subcutaneous sclerosis and keratosis. Amputation of extremities may have to be considered.

Because of the extensive exposure, this may be associated ARS with resultant granulocytopenia (causing opportunistic infections) and thrombocytopenia (causing hemorrhages).

Treatment: Inpatient intensive care

Topical antibacterial agents

Linoleic acid creams

Systemic anti-inflammatory, anti-proliferative, non-atrophic glucocorticoids to reduce edema.

Analgesia

Aspiration of fluid from bullae

Debridement of necrotic tissues

Necrectomy and full thickness skin graft

Amputation

Prevention and treatment of infections as per antibiotic sensitivity tests

Investigations: 7.5 MHz sonography to determine changes in thickness of the skin

Doppler ultrasound to assess damage to underlying larger vessels

Sonography to assess the depth of the lesion

MRI to detect deeper damage to underlying muscles

The percentage of the surface area of burns can be calculated by the rule of nine. The fluid and electrolyte/colloid requirements vary according to the grade/depth of burns. It is simpler to calculate the fluid requirement for partial burns wherein various formulae used in thermal burns may serve as a useful guide for maintenance of fluid and electrolyte balance. However, in case of whole body burns with concomitant ARS and also in deep burns as a result of irradiation, the hemodynamic stability needs measurement of central venous pressure (CVP), electrolytes, renal profile and associated management of ARS as mentioned earlier.

The management of radiation burns needs a team of:

a. Physician trained in handling radiation injuries
b. Health physicist to help arrive at the dose received and to ascertain the type of radiation (β, γ) using various dosimetric techniques
c. Hematologist (for management of ARS) when radiation burns are associated with partial or whole body exposure.
d. Plastic and reconstructive surgeon

Unlike the thermal, chemical or other forms of burns injuries, there are no physical symptoms at the time of exposure to radiation sources. The full extent of tissue damage can take a few days, weeks and even years to evolve and hence early decision regarding the type of surgical intervention has to be avoided. The only early intervention would be in the form of maintenance of fluid, electrolyte balance and life saving measures.

SURGICAL MANAGEMENT

Surgical management depends on:

a. Type of burns (β and γ)
b. The extent of surface area of burns
c. The depth of burns

The accident rate world over in atomic power reactors is extremely low as there is a well-structured radiation protection setup for man and machine. Most accidents leading to burns are reported from those using industrial radiography and some from radiological procedures in medical field like tele-radiotherapy because of inadvertent exposure to a naked (exposed) source and in interventional radiography due to non-adherence to periodic maintenance and calibration checks.

1st degree burns heal with conservative treatment spontaneously just as in the case of thermal burns. However, the recovery may take a longer time. Extensive 2nd degree burns may require split thickness skin grafting to reduce the morbidity and to expedite healing. 3rd or 4th degree burns (γ, X-rays) require investigations directed at the vascular viability of the affected tissues and procedures such as rotation flap, myocutaneous and neurovascular flaps have to be employed. Deep burns adjacent to torso and limbs have a potential to cause associated injury to hollow viscera (bladder, intestines etc.) and to the underlying bone giving rise to radionecrosis requiring additional major surgical procedures for fecal and urinary fistulae thus carrying a high mortality and poor outcome. Special mention must be made about hand injury (especially injury to digits) as this is a common form of accident. Having a hand surgeon would be a vital addition to the list of experts to help in the reconstruction treatment and rehabilitation. A timely decision regarding partial or total amputation would reduce morbidity in those injured.

Even after recovery, a possibility exists for development of late sequelae like radiation fibrosis and squamous cell carcinoma of the skin.

RADIATION PROTECTION PROGRAMS

Radiation protection involves restriction of exposure or potential exposure. This may be achieved by:

a. Engineering controls employing reducing the time of operation, increasing the distance from the source by use of remote handling operations and provision of good shielding and suitable containment to prevent the spread of unsealed radioactive materials to restrict external exposure.
b. Administrative methods such as documented procedures, good working practices, adequate supervision and restricted exposure times
c. Personal protective equipment such as lead aprons for work with X-rays, laboratory wear and industrial suits to protect against body contamination and devices such as respiratory protective equipment.

The radiation protection program works on the philosophy of ALARA (As Low As Reasonably Achievable) and contributes by preventing accidents which cause acute exposures with resultant deterministic effects and also by reducing routine exposures and the risks of stochastic effects.

Personal dosimeters are worn by individual employees and the actual doses received are measured. Personal air samplers (PAS) can measure the air activity that might contribute to an internal dose. Organ and whole body monitoring for internal exposure and biological assessments of intakes of radioactivity are used to determine internal doses.

It is important to note that everybody who deals with intervention radiological and therapeutic procedures, e.g. those undertaken by urologist, cardiologist or orthopeadicians should compulsorily use TLD (Thermoluminescent protection dosimeter) badges and stay out of the field of radiation as far as possible to prevent delayed radiation response.

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Chapter

24

Friction Burns

Karoon Agrawal

INTRODUCTION

Historically fire was produced by friction between two stones. The Olympic lamp was lit by rubbing stones and this tradition is still followed. Friction can generate a large amount of heat, so much so that it can melt stones and burn meteors in space. When rockets and space ships enter the earth's atmosphere, there is a danger of their burning due to the extreme heat produced by friction with the atmospheric air.[1] The greatly talked about Discovery space shuttle accident in which the India born astronaut Kalpana Chawla was killed along with other colleagues, is an example of the high magnitude of heat generated due to friction in space.

Burns due to friction are one of the commonest non-thermal burns. Very often, they are not reported and are overlooked in our practice as well as in literature. This is probably because in the majority of patients friction causes partial thickness burn in a small body surface area which is ignored. Usually, it is associated with mechanical injury which may be more severe than the burns. Whenever there is friction between two objects, the relatively delicate object bears the brunt of the mechanical trauma in the form of abrasion, laceration or avulsion. Hence most often the burn takes a back seat and the mechanical injury becomes the priority.

PATHOGENESIS

Friction is generated as mechanical energy when two surfaces rub against one another, out of which at least one is moving. The kinetic energy is converted into thermal energy or heat. When the delicate body surface comes in contact with this heat energy, it causes burn. As it is always associated with kinetic energy, friction burn and mechanical injury are seen together in various combinations and proportions.

The quantity of energy produced following friction is determined by the coefficient of friction which depends mainly upon the material of the surface causing friction, for example, stainless steel has a very low friction coefficient as compared to rubber on a pavement. Similarly when a part of the human body rubs against any hard object, the high coefficient of friction produces a large amount of heat energy causing body surface burn.

EPIDEMIOLOGY

Epidemiological studies of friction burns are difficult. Majority of these studies available in literature are hospital based, which have their inherent deficiencies. To know the correct incidence, community based studies are required which are neither available, nor feasible. Trying to quantitate the reported cases of friction burn is only the tip of the iceberg. A vast majority of these cases are ignored or managed with first aid or household care. Only relatively major burns or burns associated with other trauma report to the hospital, hence the fallacy of the reported incidence and degree of burn reported due to friction.

In a retrospective epidemiological study on childhood burns conducted in New Zealand in1981, 14% children had friction burns and it was the third commonest burn after scalds (40%) and contact burns (20%).[2]

In an epidemiological study from Ahmedabad, India, 60 patients of friction burns were reported over a period of two years. Majority of the patients were males in 21-30 years age group. Lower limb was affected in the majority of patients due to road side accidents. Split thickness skin graft was required in 41.7% and a flap cover was performed in 10% of patients.[3]

In an American review of 1211 children younger than 6 years over a period of 6 years there were 48 patients (4%) with friction burns because of household treadmill (Fig. 24.1). Palmar surface of the hand was the most common site.[4] Maguina et al reported 10 patients of treadmill related friction burn over a period of 4 years.[5]

In a similar retrospective study from Korea, records of 25 children with treadmill-induced friction burn were analysed. Friction burns constituted 1.4% of total number of pediatric burn injury. All lesions affected the hands and forearm. Majority affected the volar surface.[6]

The database of patients over 20 years from Brisbane and Queensland was analyzed. Out of 4523 patients admitted in the hospital, friction burn constituted 1%.[7]

ETIOLOGY

A friction burn is a non-conventional burn injury and is not reported to the hospital unless it is unusually large or else associated with other injuries. The etiological factors are very interesting and worth enlisting here (Table 24.1).

In an Indian epidemiological study of 60 friction burn patients, the Road Traffic Accidents constituted 93% and the rest were equally contributed by fall and sports.[3]

Sports activities are the commonest cause of friction burn. These are usually small, superficial and localized injuries which are mostly managed on the spot and hence, not recorded in hospital statistics. It can occur while skidding over a synthetic turf. Rope seems to be a common etiological factor in rope based sports like rope climbing, water skidding, tug of war, etc. Running causes burn over soles especially, if one runs bare-foot or with ill-fitting shoes. Aerobic dancing causes burns over the soles. The water slides in water sports cause burn due to friction against the railing and the floor of the slides, especially if the flow of water is inadequate.[8]

Table 24.1: List of etiological factors of friction burn

Sports	Skidding over turf Rope based sports[8] Bare foot trekking[23] Water slides, Aerobic dancing
Road Traffic Accidents	Car-tyre friction[12] Bicycle spoke injuries[13] Air bag friction[15-17] Loose clothing getting caught in automobile[22]
House hold and industrial machines	Treadmill[4-7,9-11] • Child activates the machine • Accidentally an unsupervised child comes in contact with the moving belt Washing machine[2] Domestic vacuum cleaner[17,18] Rolling belt in industries and agriculture
Medical equipments	Tourniquet applied over bare skin[20] Reaming of bone[21]
Miscellaneous	As a part of pathomimia[24]

Fig. 24.1: A photograph of a household treadmill for exercising

Increased popularity of physical fitness in modern society has brought many mechanized equipments into our homes. Presence of such equipment at home increases the hazard for friction burn of the hands in young children.[4-7, 9-11] A review of 1211 pediatric patients of less than six years from USA revealed treadmill friction injuries in 48 (4%) patients. The mean burnt area reported was 0.5%, mostly over the volar surface of the hand.[4] In another study, 14 injuries in nine patients were recorded. The average age of the children was 3-4 years.[10] An American study reported six hands Treadmill related friction burns at an average age of 31 months. These were mostly on the palmar aspect of the fingers.[9]

Till now all the friction burn reports were from American centers. In a retrospective study, treadmill injury from Seoul, Korea constituted 1.4% of all the pediatric burn admissions. The mean age was 3.9 ± 3.2 years. [9] This indicates the spread of treadmill related pediatric friction burn to other countries.

There are two ways by which a child can get friction burn due to treadmills at home: one is when a child activates the machine in the absence of an adult and secondly when a child accidentally comes in contact with the moving belt of the treadmill causing trauma and friction.[10]

Road side accidents are the cause of a large number of friction burns especially in hospital based studies. Al-Qattam (2000) reported 25 cases of car-tyre friction injuries of foot in children. These injuries occurred while trying to stop the car.[12] In an Indian study (2006) the incidence of friction burn reported due to Road Traffic Accidents was as high as 93.33 %[3] (Fig. 24.2).

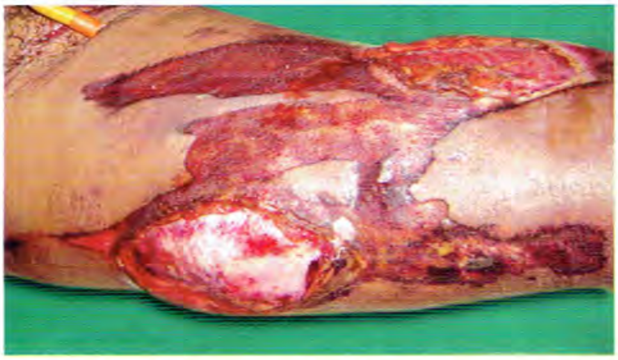
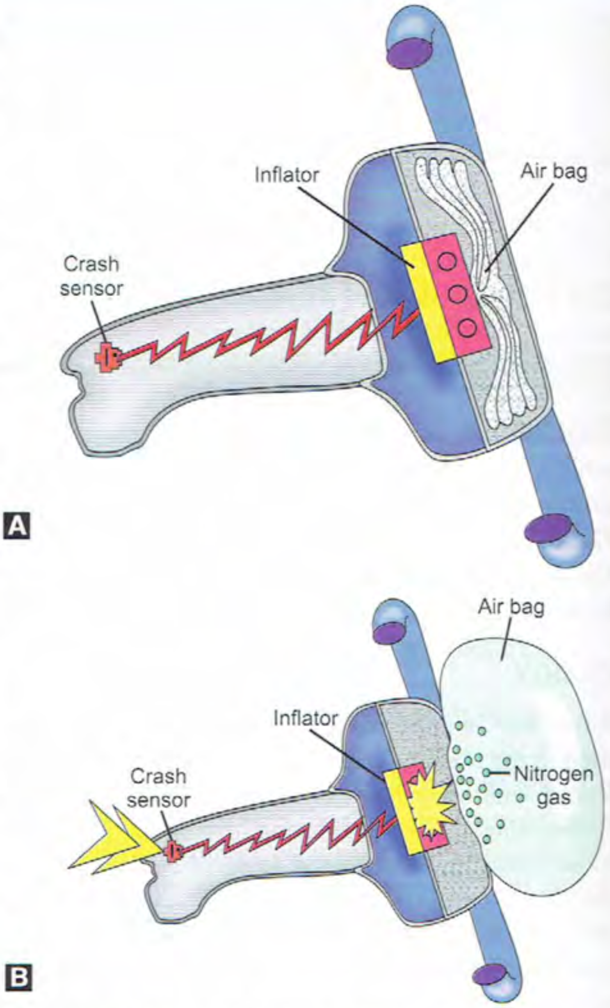


Fig. 24.2: A large full thickness burn over thigh following friction with the truck tyre. The presence of thick eschar indicates the occurrence of deep burn due to friction. There is evidence of avulsion injury laterally

In a Japanese study, 26% of their study patients of lower limb trauma caused by bicycle spoke injuries were abrasion and friction burn from the shearing effects of the spokes causing partial to full-thickness skin defect.[13] Occasionally the friction burn may be severe enough to cause full thickness loss of chest wall.[14]

Modern technology brings some unexpected problems with it. Airbags are effective and a well accepted safety device to reduce the impact of motor vehicle accidents and morbidity and mortality associated with it. Rapid deceleration due to an impact causes the ignition of sodium azide cartridge, thereby releasing nitrogen to inflate the nylon rubber bag. Most of the airbags inflate in 0.01 second at a speed of >160 km/hour[15] (Figs 24.3A and B). The physical contact with rapidly inflating airbags causes friction burn. Also direct thermal burns from high temperature gases, indirect injuries due to the melting of clothing or chemical burns due to direct contact are possible with airbags.[16,17]



Figs 24.3A and B: A line diagram showing the inflation of car airbag after impact. The airbag inflates with nitrogen after the crash sensor activates the inflator

There are many other interesting etiological factors causing friction burn reported in literature. These are caused by common machines used in day-to-day life and are commonly ignored. There are reports of friction burn caused by washing machine wringers[2] and domestic vacuum cleaners[18,19] (Fig. 24.4). Among the medical equipments, tourniquet applied over the thigh without padding causes friction burn[20] and the reaming of bone significantly increases the temperature inside the bone. Rolling belts in industries are hazardous as these cause friction burns.[21]

Loose clothes around the body may get caught in a revolving machine or in automobiles and can snare the neck or other parts of the body. In a freak accident, a 17-year-old girl had friction burn over the neck due to her 'Chunni' (also called, 'dupatta'), a traditional Indian clothing, wrapped round the neck caught in a three wheeler auto wheel.[22] Bare Foot Mountain trekking can cause friction/frost burn.[23]

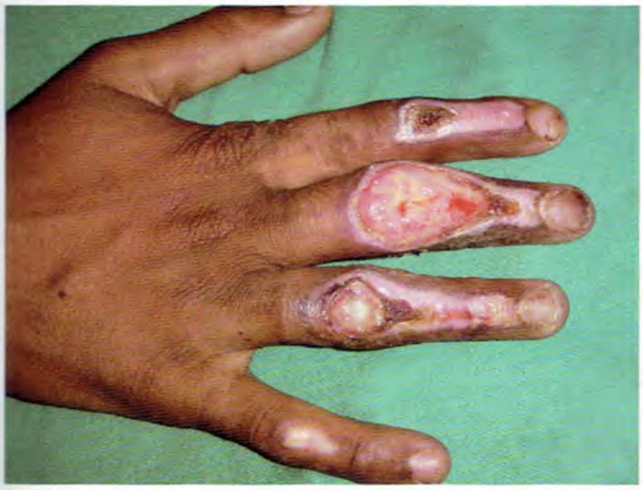


Fig. 24.4: Friction burns over dorsum of fingers because of hand coming in the pressing machine, showing full thickness loss in the center surrounded by zone of partial thickness skin loss

Friction blisters may be a manifestation of pathomimia, which can be produced by nails, razor, blades, cigarettes, chemicals etc. These are not true friction burns, but resemble them; hence, we should be aware of them.[24]

Apart from these etiological factors environmental factors play a very important role in the occurrence of and in determining the degree of friction burn. In summer season the incidence and magnitude of friction burns is more. Inadequate clothing in summer also increases the risk. Pegg and Mayzee (1982) have pointed out that only 9% of friction burns occurred during winters, due to adequate clothing. On the other hand, the incidence was much more in summer because of inadequate clothing like use of shorts and thin flimsy shirts.[7]

TYPE OF BURNS

The burns inflicted with friction are mostly minor and superficial (Figs 24.3 and 24.4) However, there are a few reports of deeper friction burns too.[3,14] In an Indian study of sixty burn patients 31.66% had first degree (Fig. 24.5), 20% had second degree (Figs 24.6 to 24.8C) and 48.33 % had third degree burns [3] (Figs 24.9 and 24.10). This is a hospital based study; hence, a large number of more severe burns. Majority of small, superficial first and second degree burns may have taken treatment at home or from private clinics and hence, not reported.

Majority of the accidental friction burns in children take place at home. Although the burn is small, it should be taken seriously in children.

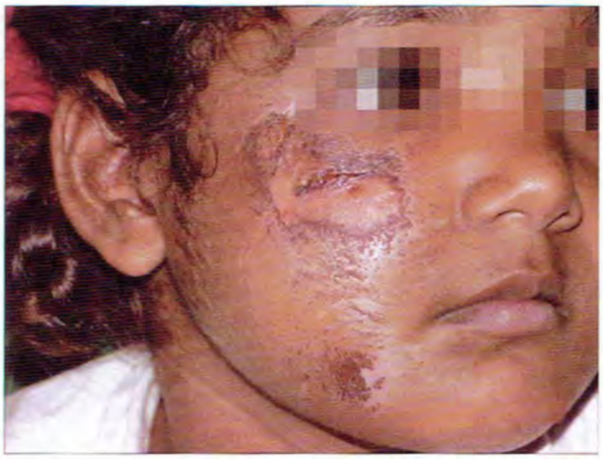


Fig. 24.5: A typical superficial friction burn over face after road side accident in a child

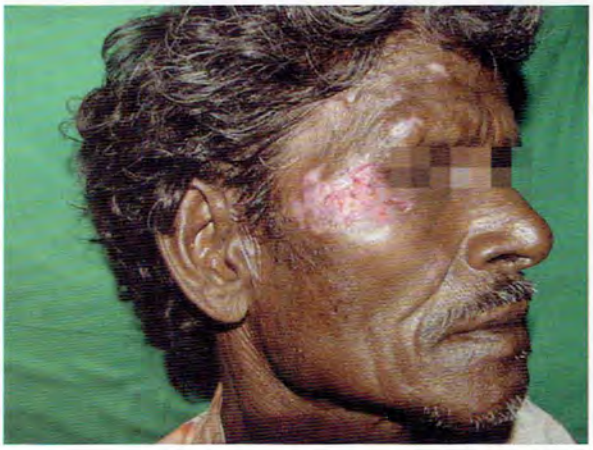


Fig. 24.6: A deep partial thickness friction burn over the right zygomatic region following road side accident requiring closure of wound by grafting

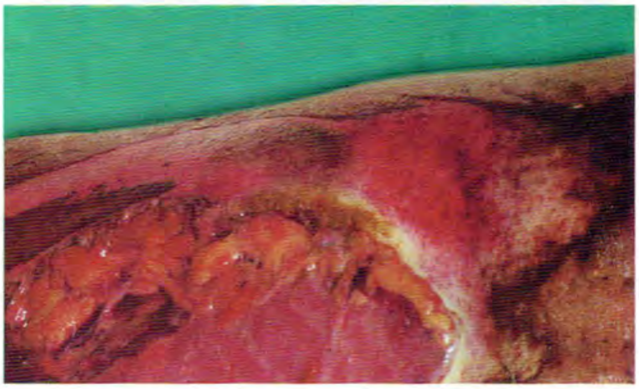


Fig. 24.7: Deep friction burns with associated avulsion injury in thigh of a young man following road traffic accident

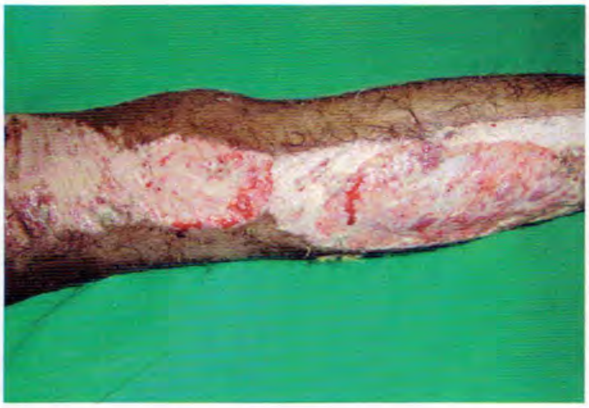


Fig. 24.8A: Typical friction burn over lower limb because of truck tyre running over the limb

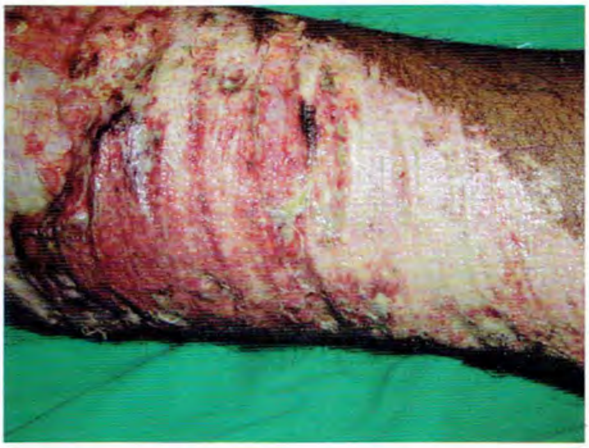


Fig. 24.8B: Same case showing the tyre marks with intermixed superficial and deep dermal burn with total loss of skin in the proximal thigh (extreme left)

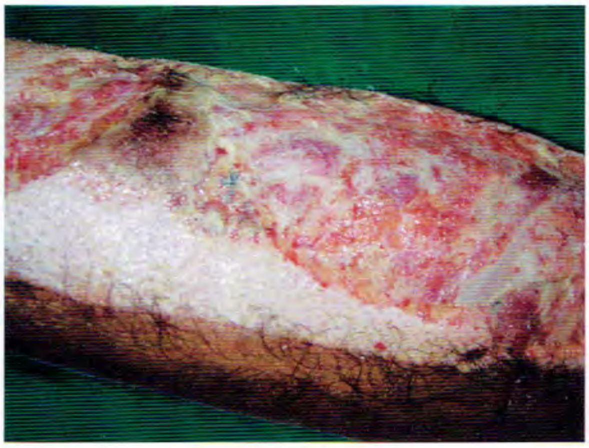


Fig. 24.8C: Close up view after 4 days showing some remanant of dermis in the center with superficial burn in the periphery in healing phase, requiring skin grafting in the center

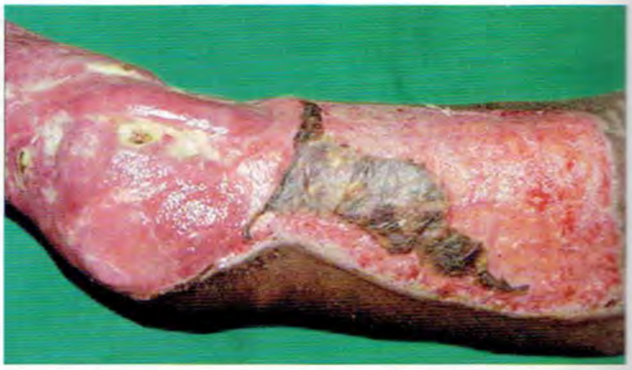


Fig. 24.9: A large full thickness burn over thigh following friction with truck tyre. The presence of thick eschar indicates deep burn due to friction. There is evidence of avulsion distally

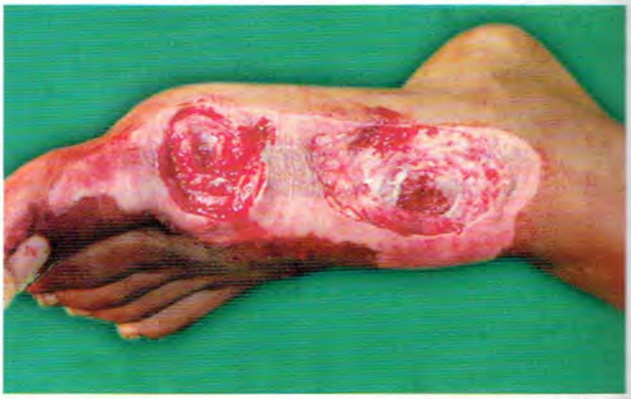


Fig. 24.10: Full thickness friction burn over dorsum of foot by car tyre running over it

MANAGEMENT

It is simple and straight forward because of the limited extent of burn in the majority of patients. The principles of management of friction burns are the same as for thermal burns. Majority of the friction burns are managed as minor burn with tetanus prophylaxis, local wound care, systemic antibiotics and analgesics. Sometimes, it requires surgical management with tangential excision and split thickness skin grafting. In treadmill related injuries 46% of children with friction burn had full thickness burn requiring surgical management.[5] In another study 33% of Treadmill related friction burn cases required skin grafting.[9] Major friction burns are associated with other injuries which should be managed simultaneously. In an Indian study 35% had hand fractures, 10% had head injury and 5% had associated tendon and nerve injury.[3]

Majority of the airbag friction burns are minor and partial thickness and do not require hospitalization.[17] Though the majority of airbag lesions are minor, correct diagnosis of type of burn is necessary as it may be a friction or chemical burn or mechanical trauma. Based on the type of trauma, the appropriate treatment is instituted. The benefit of airbags far outweighs the risks of the burns and hence are acceptable.

In the Indian study, 41.66% required split thickness skin graft (Fig. 24.11) and 10% required major flap procedures[3] (Figs 24.12A to C). A full thickness defect of the chest-wall has been reported requiring multiple flap reconstructions.[14]

Early recognition and appropriate management result in excellent functional outcome with minimum morbidity.

PREVENTION

Majority of unreported and reported friction burns occur during sports and routine activities and hence, these may not be totally preventable in a large number of cases. However, appropriate preventive habits, prevention of road side accidents and specific precautions observed while dealing with etiological factors may reduce the incidence of friction burn.

Benever et al predict that the incidence of friction burn injury with treadmill like domestic machines is likely to increase in future unless protective modification of exercise machine is done.[9] Additional safety measures, warning labels and parental education can prevent friction burns due to domestic and industrial causes.

Proper use of seat belts through shoulder and lap reduces the overall injury to the car riders. The airbag friction burn is also less when the riders have their seat belts on.

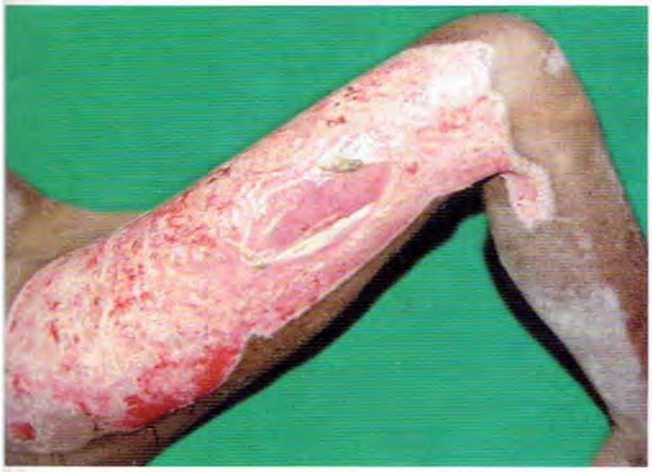


Fig. 24.11: Combination of friction burn and avulsion due to road side accident. The Proximal and distal parts of the raw areas are following the separation of eschar. Central area with exposed muscle is due to associated avulsion injury. These areas can be covered with split skin graft alone

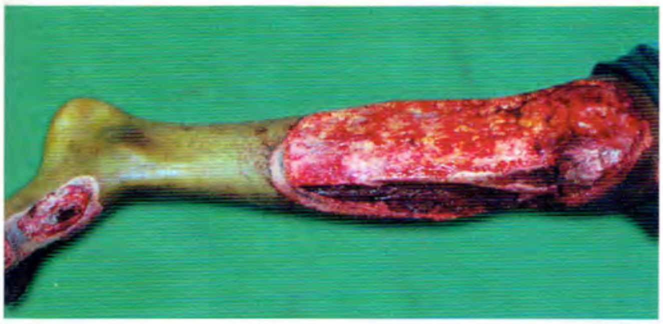


Fig. 24.12A: Exposed tibia after removal of thick eschar following deep friction burn over leg in road traffic accident

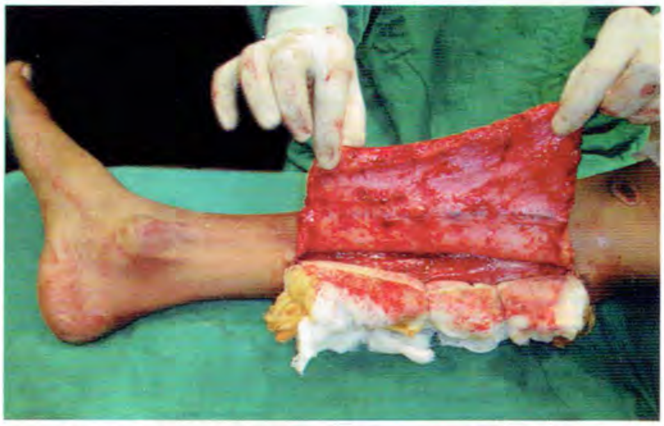


Fig. 24.12B: Fasciocutaneous flap raised from opposite leg for cover

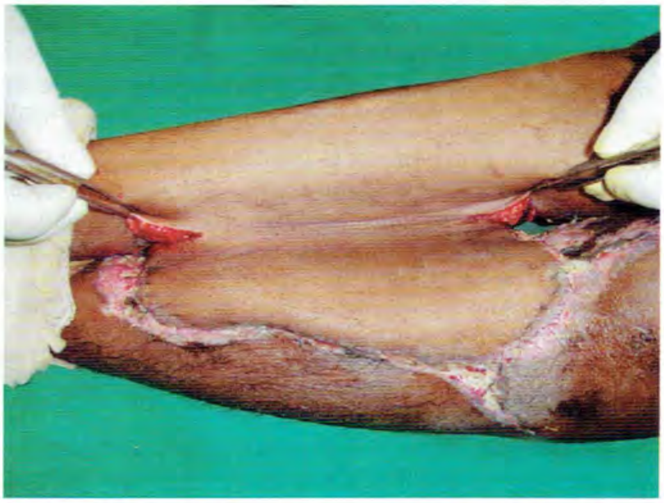


Fig. 24.12C: Inset cross leg flap covering the same defect completly

SUMMARY

To conclude, the friction burn is an interesting trauma associated with day-to-day active life. One has to accept the occurrence of friction burn as a part of life. Modern gadgets have become a part of today's lifestyle, which may increase the possibilities of friction burn. There are a few areas where preventive measures and watchful lifestyle may significantly reduce the incidence of friction burn.

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Chapter

25

Firework and Firecracker Injuries

Arun Goel, Sameek Bhattacharya

INTRODUCTION AND HISTORY

Fire, which changed the face of human civilization, has been man's fascination since its discovery. Among the modalities of its use, ranging from highly beneficial to highly destructive, fire has been central to numerous occasions of celebration. Initial uses were limited to decorative illumination but later, with the evolution of ordnance technology, developed the art of manufacturing and setting off of fireworks (Pyrotechnics). The earliest documentation of "fireworks" is from China during Han dynasties (200 BC) which dates even before the discovery of gunpowder. Burning of green bamboo led to expansion of trapped air resulting in loud bang. The bursting bamboo or "pao chuk" in Chinese, were part of Lunar New Year celebrations and other festive moments for about a thousand years till gunpowder was invented during Sui and Tang dynasties (600-900 AD). The precursor to the discovery of gunpowder was a chance finding of a sulfurous mixture by Chinese alchemists during the course of their experiments, which burst into illuminating flame on burning. "Hao yao" or fire dung as they called it were packed in bamboo tubes and burned as firecrackers. The knowledge of pyrotechnics reached the West only in 1200 AD when Marco Polo brought the Chinese know-how to Italy. It was the Italians who added the esthetic dimension to the fireworks by adding metal powder to gunpowder in order to produce colorful sparkles. With further advances in chemistry, some more metal salts were added to produce multi-colored pyrotechnic-effects.

The new age pyrotechnic devices are broadly classified as incendiary fireworks and explosive firecrackers. A firework is a low explosive pyrotechnic device used primarily for esthetic illumination and entertainment in the form of light, sparkles and floaters, noise being secondary. A firecracker, on the other hand, is a relatively higher explosive device designed primarily to produce noise in the form of a loud bang and visual effect is incidental. Both firecracker and fireworks are basically explosives wrapped in heavy paper casing with a long fuse to ignite.

FIREWORK/FIRECRACKER SCENARIO IN MODERN TIMES

In modern times, social events like marriage, religious functions and state functions, e.g. Independence Day all involve display of fireworks and bursting of firecrackers. So much so that even fireworks competitions are being organized at a number of places in the world, for example, the annual Montreal Fireworks Festival in Canada and World Pyro Olympics held in Philippines. Many major cities around the globe begin the New Year with fireworks and firecrackers. The opening and closing ceremony of Beijing Olympics in 2008 were marked by one of the greatest firework display. The fireworks/firecrackers have also been known for their use to exorcise evil spirits with their loud sound. Firecrackers are even utilized by farmers as a modality to scare away wild animals and birds from farms.

Its use in festivities is universal all over the world, be it a developed or under-developed nation. In India, widespread and large scale use of firecrackers and fireworks during "Diwali" ('festival of lights') is unparalleled in the world. "Diwali" or "Deepawali" is celebrated to mark the victorious return of Lord Rama (an incarnation of Lord Vishnu, a Hindu

God) to his kingdom in Ayodhya after killing the evil Ravana (the demon king) of Lanka (now Sri Lanka). During this festive night of victory of good over evil, the houses are adorned with small earthen oil lamps, candles and colorful electric lights. After offering the prayers, the entire family participates in the bursting of firecrackers. Twenty days preceding "Diwali", "Dussehera" is celebrated by burning the towering effigies of Ravana, his brother Kumbhakarna and son Meghnada. These are packed with massive amounts of various types of firecrackers and fireworks and this public display is attended by thousands of onlookers in every locality. "Diwali" is also celebrated in many other countries of South East Asia having large population of Indian origin.

Thrissur Pooram, a two century old festival to mark the end of caste supremacy is celebrated in Kerala, southern India. The festivities are attended by millions of people from across the state and even international visitors. The spectacular fireworks display, biggest in India, starts in the early hours (0230 Hours IST) and lasts for 3-4 hours.

During marriages in India, firecrackers are burst and fireworks are arranged with complete disregard to safety measures. The area may be a congested, narrow road with vehicles, children surrounding the site at close range as onlookers and ladies wearing synthetic, inflammable free flowing dresses. The make-shift "pandals" (tents) are also made of inflammable cloth. In India, even victory in international cricket matches, whether played in or out of country, is celebrated by cricket fans at home by bursting crackers.

Hari Raya Puasa, marking the culmination of"Ramadan", the holy month of fasting is celebrated in Malayasia by Muslims. Shab-e-Barat in Pakistan, Independence Day in USA and Halloween season in Ireland and elsewhere are all celebrated with fireworks and firecrackers.

Presently fireworks/firecrackers are billions of dollar industry giving employment to millions from manufacturing to retail sale. China is the largest manufacturer and exporter in the world. Sivakasi city in Tamil Nadu is the leading producer in India.It has a great share in world market as well. Thus, it is clear that despite their immense harmful potential, it may not be possible to effectively ban their manufacturing and use. From time to time different nations have tried partial or total ban on them which only led to illegal manufacture and smuggling. Such illicit firecrackers, obviously devoid of standard safety features, posed greater risk ultimately lifting of ban allowing restricted use. For example, in Singapore, a partial ban on firecrackers was imposed in 1970 and converted to complete ban in 1972. However, in 2003 firecrackers were allowed to be set off during festival seasons under controlled conditions by Singapore Tourism Board. Similarly, in United States in 1973, the National Society for the Prevention of Blindness proposed to The US Consumer Product Safety Commission (responsible for regulating fireworks under Govt. Act) a total ban on the sale of fireworks for personal use and a temporary ban was imposed. Despite this ban having the support of American Academy of Pediatrics, American Public Health Association and National Fire Protection Association, etc. the ban had to be lifted the same year. At present, although firecrackers are not illegal in US, there are strict regulations regarding their sale, possession and use.

The fireworks/firecrackers are so much ingrained in the Indian psyche and socio-cultural fabric that imposition of a total/partial ban by the authorities is next to impossible. Whatever laws are in place, the slackness in their execution allows rampant violations in manufacture, storage, sale and individual use by the public leading to a large number of cracker-related burns, blast injuries, deaths and disablement apart from economic loss and loss of property. The sale of fireworks is largely unrestricted in India and people have access to a variety of fireworks including illicit and poorly standardized 'make' and light them in densely populated parts of cities and towns without a hint of safety concern. Improper use of fireworks may be dangerous, both to the person operating them as well as to the bystander(s).

TYPES OF FIRECRACKERS

The various types of firecrackers available are:

1. Phooljhari or "Sparkler" (Fig. 25. 1): This is commonly used in lighting most of the other firecrackers. It consists of a metal stick with one-half covered with pyrotechnic compounds and the other half acts as a handle.
2. Anar or "fountain" or "flowerpetal" (Fig. 25.2). It emerges as a big sparkling fountain of fire when lit. In addition, "Anar" is unique to our country and many people make its crude form at home, which poses grave risk while preparing as well as igniting them.
3. Zameen Chakri or Ground spinner (Fig. 25.3): It is a spinning circle of fire with sparks coming out of it.
4. Rocket (Fig. 25.4A): When fired, goes in the sky and bursts into myriad of colors and designs. They may prove dangerous when they change their course and enter into residential buildings. In addition, they may start fires after landing on inflammable material.

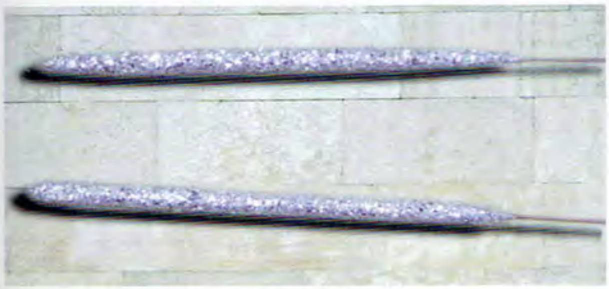


Fig. 25.1: Phooljhari

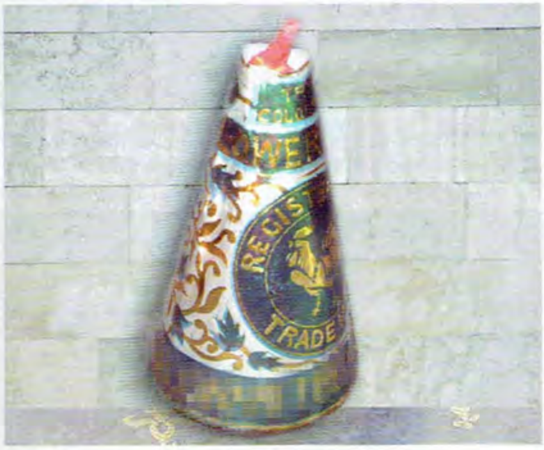


Fig. 25.2: Anar

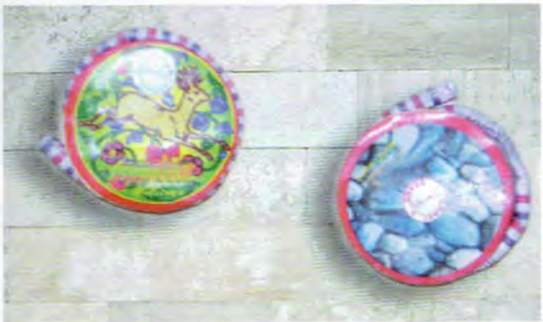


Fig. 25.3: Zameen chakri

5. Bombs (Figs 25.4B to D) are available in various sizes and intensity. On bursting, they produce a lot of noise. The largest ones may even rupture the ear drum due to their intense blast wave. Many people tie a large number of them in series so that the noise continues for some time in multiple outbursts till all the bombs explode. Some others ignite them in their hand and throw away just before it explodes. All these are highly dangerous practices leading to a lot of injuries.

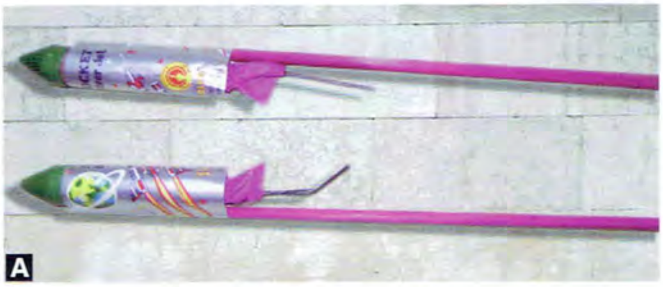
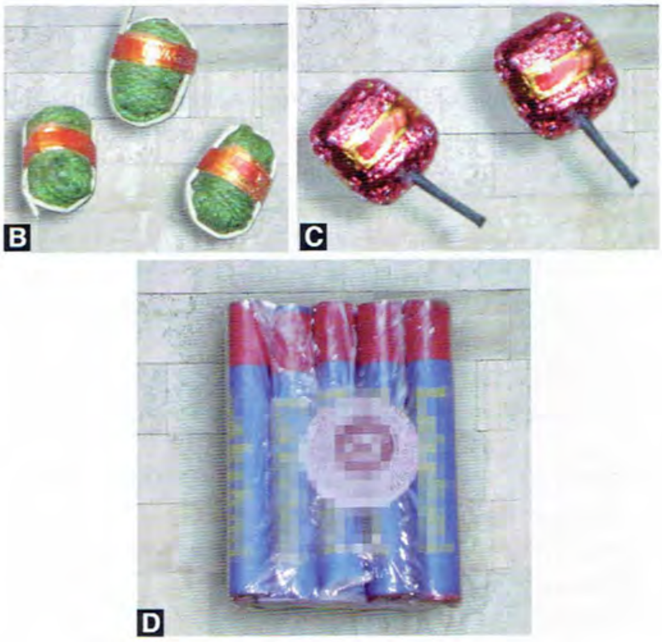


Fig. 25.4A: Rocket



Figs 25.4B to D: Bombs

6. Miscellaneous: "Snake" consists of an innocuous looking small black tablet which when ignited enlarges in the form of a long snake emitting a lot of smoke.

EPIDEMIOLOGY

The epidemiological profile of firework injury is unique to a region and culture and intimately linked to education, economics and legalities. In the US the number of fire cracker-related cases has dropped from 10,900 in 1991 to 5,700 in 2007. Although no national data is available for India, the figures must be far higher considering the magnitude of their use almost whole of the year, larger population, scant safety consciousness and poor regard for abiding by the laws. By virtue of their inherent aggressiveness, the males handle fireworks/firecrackers more and account for majority of victims. Most of the victims are children below 15 years of age and young adults. In US the firecrackers followed by sparklers are the leading causes of firework injury. In India, especially in Delhi, "anar" has been incriminated in majority of cases followed by other firecrackers.[1] A study from Ahmedabad reports firecrackers followed by lighting of powder taken out from firecrackers, rockets and the 'anar' as the commonest causes.[2]

Fireworks/Firecrackers cause injuries and damage in the following circumstances:

1. During production: To manufacture fireworks/firecrackers, one has to obtain a license from the governmental authorities and comply with the rules and regulations laid therein. Unfortunately in India, there is mushrooming of illegal factories even in residential and thickly populated areas. Safety norms are flouted. When these factories catch fire, there is not only loss of life but the adjoining houses also catch fire and collapse with loss of property. Once these poor quality products reach the hands of general public, they frequently malfunction leading to burns and other injuries.
2. Storage: Once out of factory, these products, whether produced under license or not, are stored illegally in thickly populated and unlicensed areas and godowns with great risk of catching fire.
3. Retail sale: During festive season like Diwali, a number of shops come up in every locality selling crackers in open spaces as if they are innocuous items, again endangering the shoppers.
4. Improper and unsafe use of fireworks/firecrackers by end users (Figs 25.5A and B):
   a. Simultaneous igniting of multiple crackers/sparklers.
   b. Igniting 'bombs' and 'anar' in hand in an act of bravado.
   c. Bowing over a firecracker while trying to light it.
   d. Unsupervised bursting of firecrackers by small children.
   e. Trying to find out the cause of non-bursting of a cracker in expected time or relighting it.
   f. Collection of the combustible powder out of a number of crackers and burning them in a heap.
   g. Throwing lighted crackers on onlookers or a passerby.
   h. Firecrackers exploding earlier or later than expected.
   i. Errant flight paths of 'rocket' and spinner.
   j. Sparks or debris from crackers/fireworks causing house fire.
   k. Trying to make crude crackers (e.g., 'anar') at home.
   l. Bursting cheap quality crackers.
   m. Insufficient open space.

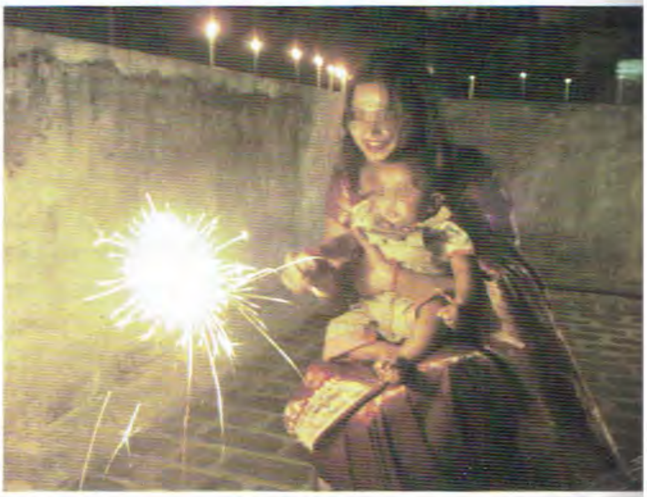


Fig. 25.5A: Unsafe use of 'Phooljari' by adults wearing synthetic clothes and holding a child in the arms

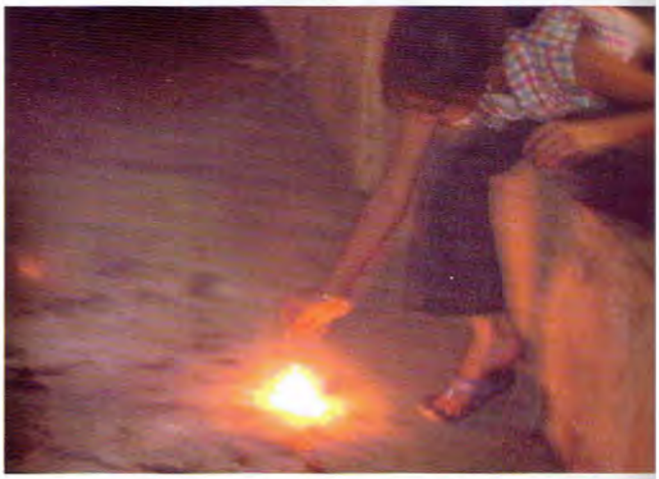


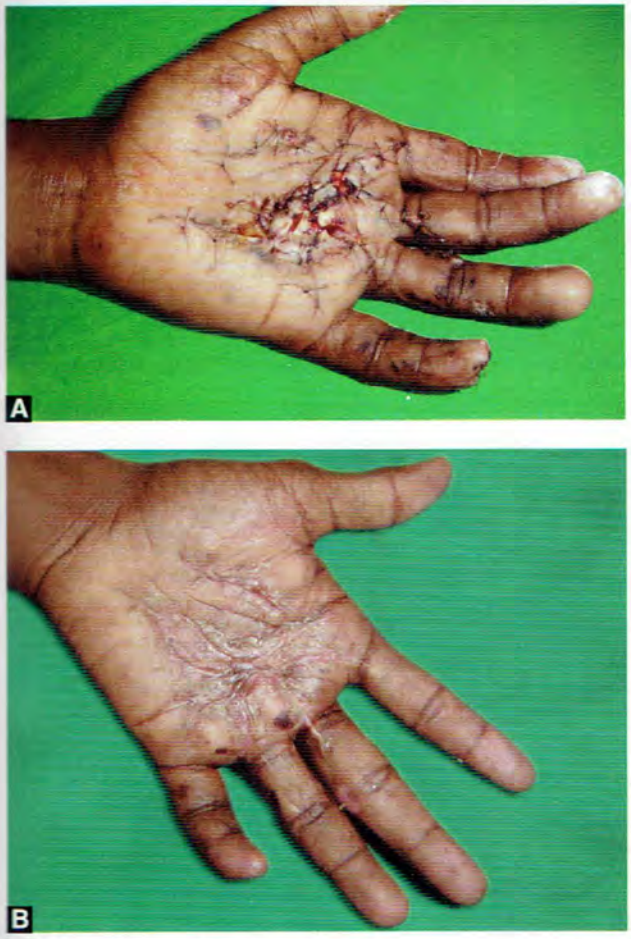
Fig. 25.5B: Unsafe use of 'Zameen Chakri' by a child who is likely to get burns of feet

CLINICAL FEATURES OF FIRECRACKERS/FIREWORKS INJURY

The fireworks/firecracker related injuries[3-8] can be divided into various types, namely, burns, injuries of the ocular globe and adnexa, lacerations, amputations, noise pollution and deafness and respiratory illnesses produced/precipitated by smoke pollution in the environment, etc.

Most of the burn injuries due to firecrackers affect the hands (Figs 25.6 to 25.10), face (Figs 25.11 to 25.15) and eyes. However, any part of the body may get burnt especially when clothes catch fire due to crackers. The feet may get burnt by stepping on the hot sparkles from 'phooljhari' or 'Zameen chakri' (Fig. 25.5B). The injuries can be due to flash, flame, contact and blast. The type of injury varies from place to place depending on the prevalent type of cracker being used more often. Thus, in Italy, lacerations and amputations of hand are more common because explosive devices are used more commonly. In India and USA, burns are more common. Flash burns are usually superficial and caused by crackers like 'anar', 'charkhi' and sparklers affecting bystanders as well.

Facial burns can be complicated by injury to eyeball and ocular adnexa and account for some of the most disabling firecracker- related injuries.[9-13] They may be penetrating or non-penetrating. They include eyelid burn/laceration/hematoma, conjunctival burn/contusion/foreign body and corneal burn/contusion/foreign body, corneal perforation or hyphema, traumatic cataract, rupture of iris sphincter or even globe, vitreous/macular hemorrhage and retinal edema, etc. There may be a partial or total loss of vision. Most of these injuries are caused by rockets fired from a bottle.



Figs 25.6A and B: Explosion of 'Anar' in hand causing (A) laceration with burns with stitched wounds. (B) Healed

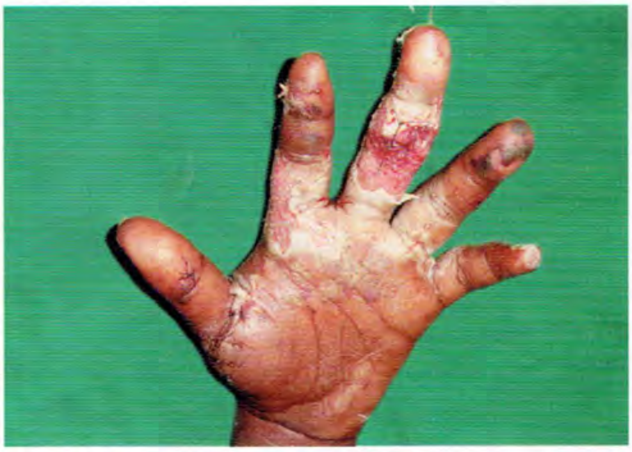


Fig. 25.7: Laceration of first web space with burns

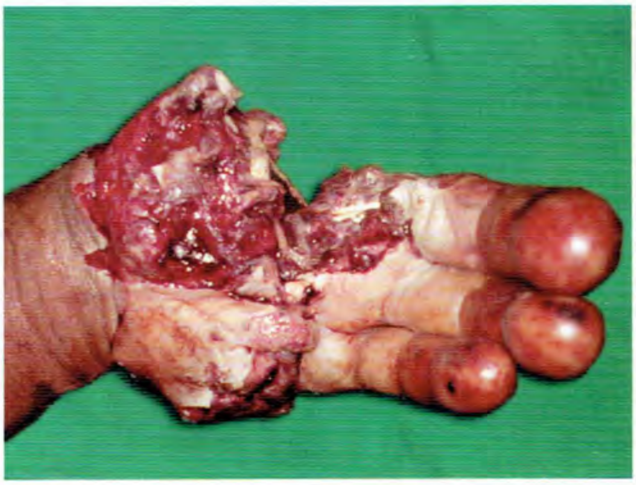
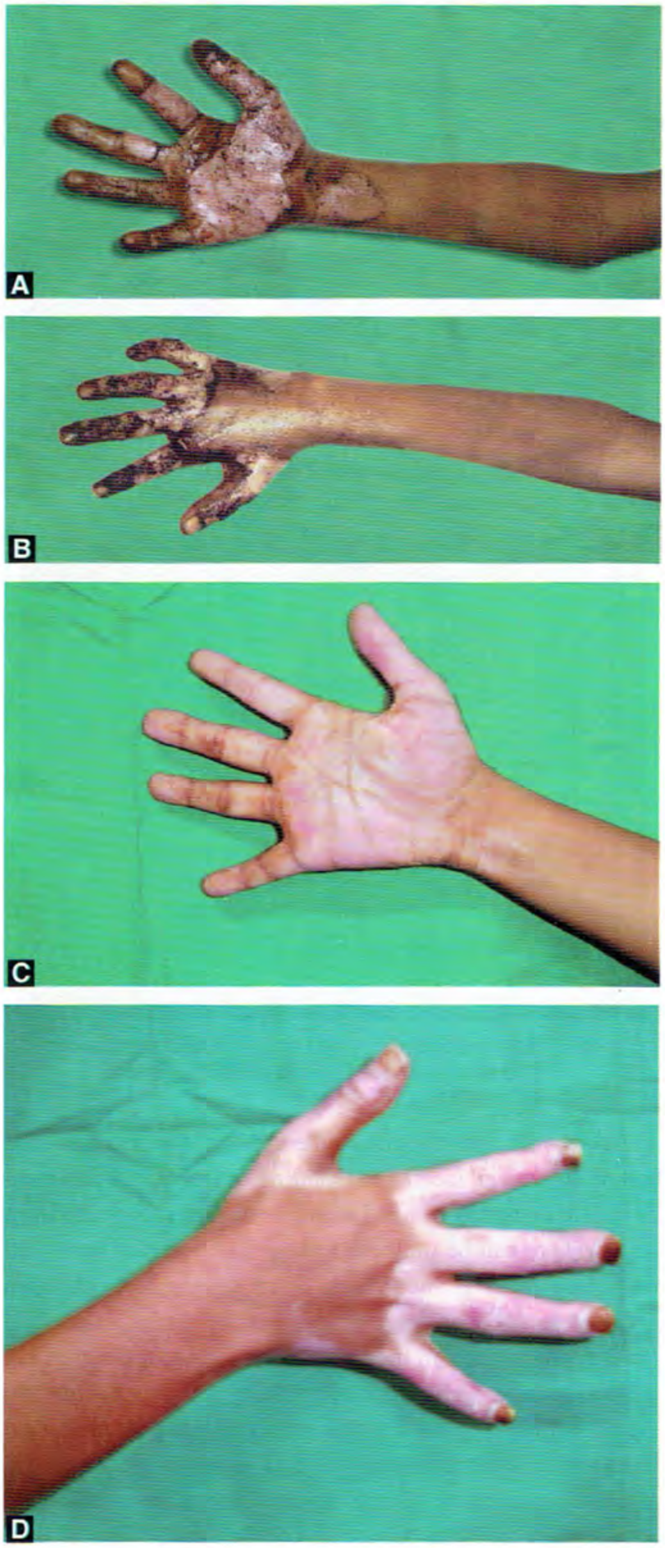
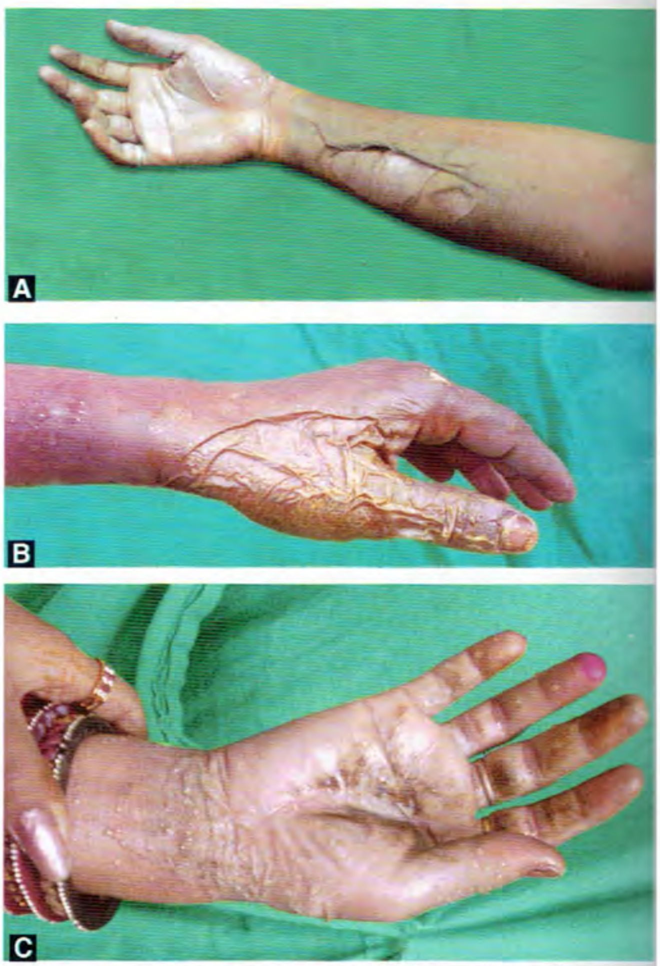


Fig. 25.8: Massive lacerations with avulsions and amputations of thumb and finger due to bomb exploding in hand

Blast injury mainly affects the hands of the operators and can be contusion, laceration, splitting of web space and even complete mangling of hand with loss of digits (Figs 25.6A to 25.8). There can be multiple fractures of bones of hand and even loss of skin and bones. Such severe blast injuries afflicted to the operator are mostly by powerful bombs and 'anar', which burst in the hand. The wounds are extremely painful as white phosphorus particles are driven into the skin due to blast effect. These foreign bodies later on lead to tattooing after healing. The self propelling rockets are also known to cause severe damage if they are misdirected accidentally and can be responsible for major fires if they land up on an inflammable material instead of going up in the air and bursting in the sky. Flame burn injuries due to crackers may be extensive if clothes catch fire.

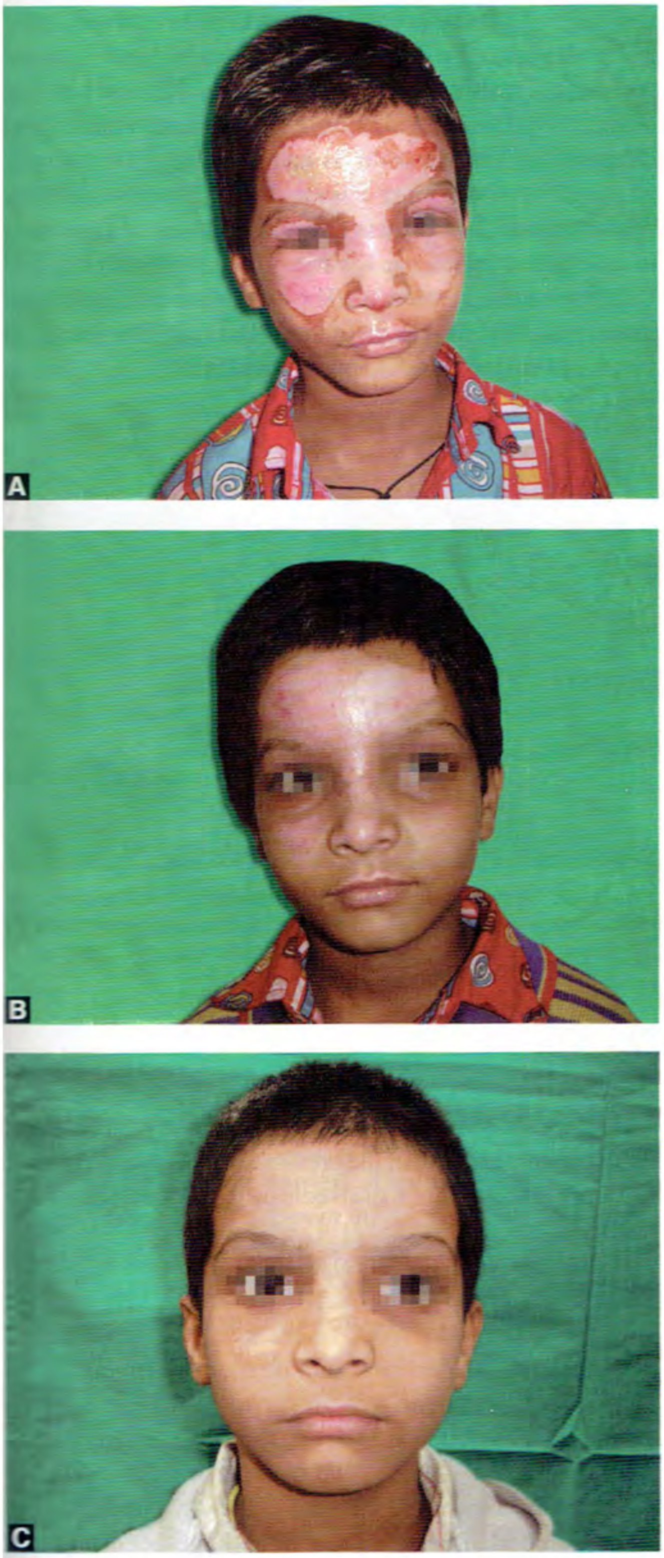


Figs 25.9A to D: Burn due to cracker. Healed after conservative treatment. Please note the application of "Ratanjyot" (indigenous medicine) and a "sacred thread" tied on the wrist used in some parts of our country for faster healing



Figs 25.10A to C: Burns due to firecrackers

In addition to fire-related mishaps, the fireworks/firecrackers also cause noise and air pollution. Infants and small children do get frightened on hearing the loud sounds produced.[14] Elderly and ill may also not be comfortable with them. In some bombs, the sound level produced is more than100 decibels. This is known to cause damage to the tympanic membrane leading to temporary deafness which recovers in 7-10 days. If the sound level is more than 140 decibels, permanent deafness can result. To avoid this, the regulations do not allow bombs with more than 80 decibels to be manufactured. A lot of smoke is produced especially on the 'Diwali' day and precipitates the attacks of asthma in susceptible people. The by-products of combustion contain various toxic and non-toxic chemicals which can cause inhalational injury. Thus, signs and symptoms of respiratory tract injury must be looked for in burns in closed spaces due to firecrackers.



Figs 25.11A to C: Superficial burns of face due to cracker injury being managed conservatively; healed

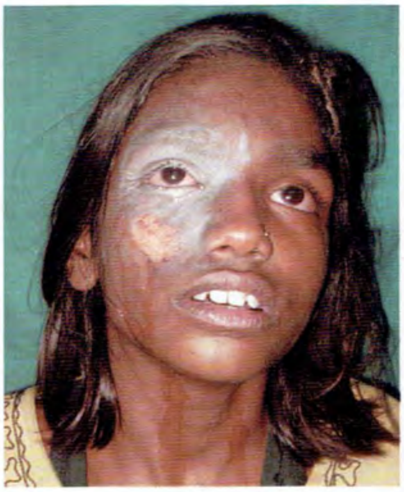


Fig. 25.12: Cracker injuries to face and right eye

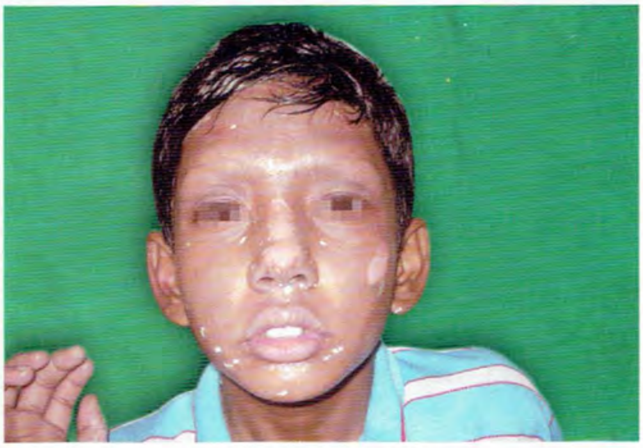


Fig. 25.13: Cracker injuries to face

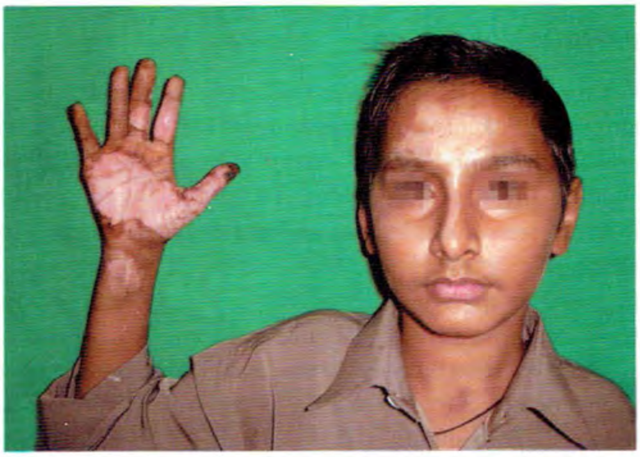
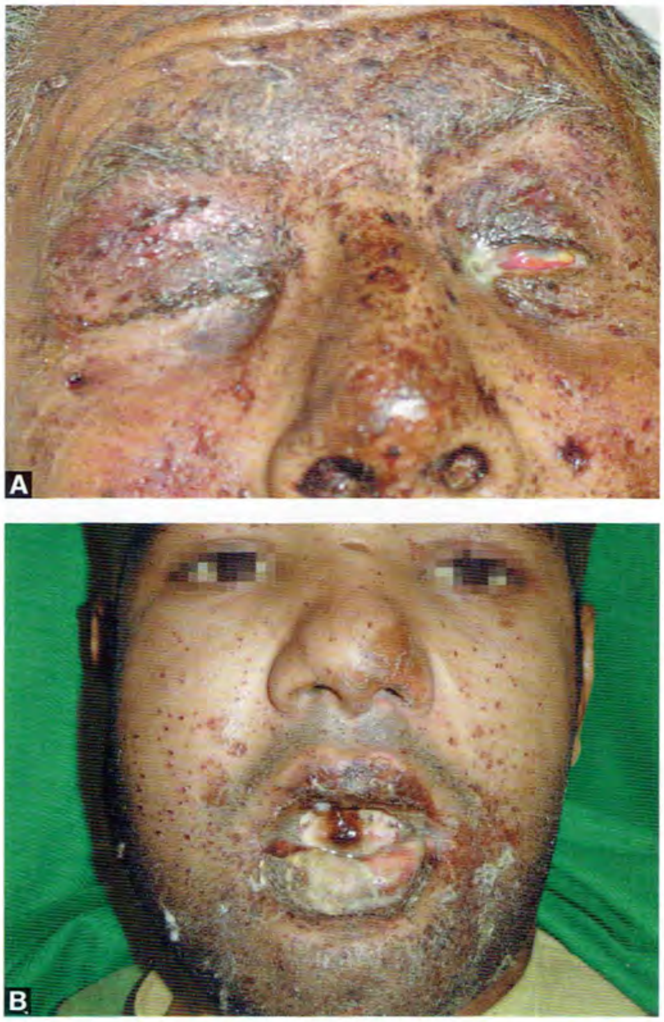


Fig. 25.14: Cracker injury to hand and face, a usual combination



Figs 25.15A and B: Cracker injuries to face with cracker powder causing tattooing

MANAGEMENT

Not more than 10% of the firecracker injuries require admission. Patients are generally treated on outpatient basis. However, the moderate to extensive burns due to clothes catching fire need to be admitted. Patients coming with ocular injuries, blast injuries of hand and facial burns with edema require admission. Management of burn wound is the same as for any other minor or major thermal burn which has been already elaborated elsewhere in this book. All burns must be thoroughly washed with saline to remove foreign bodies before dressing. Similarly in cases of ocular injuries the eyes should be thoroughly irrigated. Phosphorus particles have to be removed manually followed by saline irrigation and Fluorescein staining done for corneal involvement. The management of these injuries is beyond the realm of a burn surgeon and requires the intervention of an ophthalmologist.[15] For lacerations of the hand, suturing is done to reposition the tissues back to as normal as possible (Fig 25.16). The blast injuries of the hand need meticulous debridement under anesthesia and fixation of fractures by K-wires or rarely by mini plating and secondary reconstructive procedure(s) should be planned especially in case bones are exposed. Various loco-regional or distant flaps have to be considered. The most commonly used flap is the groin flap. Severe mutilating blast injuries of the hand may need amputations at appropriate levels.

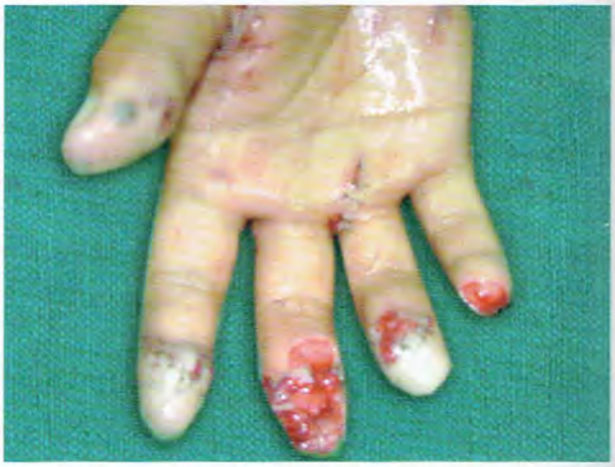


Fig. 25.16: Blast injury of hand due to firecracker leading to multiple lacerations and loss of soft tissue in the finger tips–managed by reposition of tissue and local advancement flaps and skin grafting

PREVENTION

Although firecrackers/fireworks lead to a lot of morbidity and mortality and destruction of property, a complete ban is not feasible because of the pressure of a large and growing firework industry and the socio-religious nature of the festivals.

Over the years, with sustained education of school children about the harm caused by firecrackers coupled with the slogan, say 'NO' to crackers, the incidence of these injuries has declined. In Safdarjung Hospital, Delhi, the number of patients presenting to Burns Casualty on Diwali night has fallen to 131 in 2008 from over four hundred cases two decades back (authors' personal unpublished data) despite a manifold increase in the city population.

A number of recommendations have been made to prevent fireworks/firecracker-related injuries. The most important consideration is the recognition by the general public as well as the authorities the fact that these innocuous looking toys in various shapes and sizes covered in attractive, multi-colored paper have the full potential of harm as we relate with the military combat ammunitions. Hence, the laws with respect to their manufacture, quality control, transportation, storage and retail sale must be stringent, modified and upgraded from time to time and strictly enforced. As far as possible, there should be community display of fireworks in open areas by experts and individual use of crackers by one and all should be discouraged.

A number of unsafe practices at end user level have already been enumerated above and one must avoid them. Some other precautions to be observed are listed below:

1. Ensure that other people are out of range and warned before lighting fireworks.
2. While firing rockets ensure that it is upright in a stable bottle.
3. Keep a bucket of water ready in the area where crackers are burst. In case of any mishap, pour water.
4. Wear tight fitting thick cotton dresses.
5. Store firecrackers in a safe place until they are needed, away from risk of fire.
6. Always use a long 'phooljhari' or a candle to ignite crackers keeping the elbow joint straight to maximize the distance between cracker and the body.
7. Do not apply oil, toothpaste or any other home made remedies/ointment on burnt area.
8. Do not panic and drive recklessly while transporting the victim to medical services. A delay of up to one hour does not matter.

SUMMARY

The fireworks/firecrackers are more than two millennia old and have become a source of recreation for people all over the world breaking the barriers of varied cultures, religion, literacy, urban-rural, rich-poor, developed-under developed etc. They are associated with religious festivals, national state functions, different New Year's Day of various countries, sports, marriages, etc. They are billions of dollar industry. They are also associated with large morbidity, disablement and mortality apart from economic loss and loss to property. The World Health Organization[16] called for a worldwide ban on manufacture of all fireworks to prevent blindness from this cause in 1984. Quarter of a century later in 2009, we know that their total ban is not possible. The only solution lies in their manufacture and sale as per governmental regulations, strict enforcement of laws related to them and proper precautions being observed by the end user. Various studies[17,18] conducted in different countries have demonstrated the positive effect of such measures in minimizing the 'death' and destruction commonly associated with their use. Thus, during "Dussehera-Diwali" days in India, around Independence Day in USA, New Year Day in Italy, China and everywhere else, the incidence of these injuries has certainly come down due to concerted efforts of the various governmental/non-governmental agencies, schools and media participation in teaching the public about dangers of firecracker use, using only 'safe and sane' crackers, precautions and safety measures to be taken etc.

The festivals are occasions of joy and firecrackers/fireworks are an enjoyable spectacle. They must not be turned into 'unfortunate dates' to be remembered for the rest of one's life.

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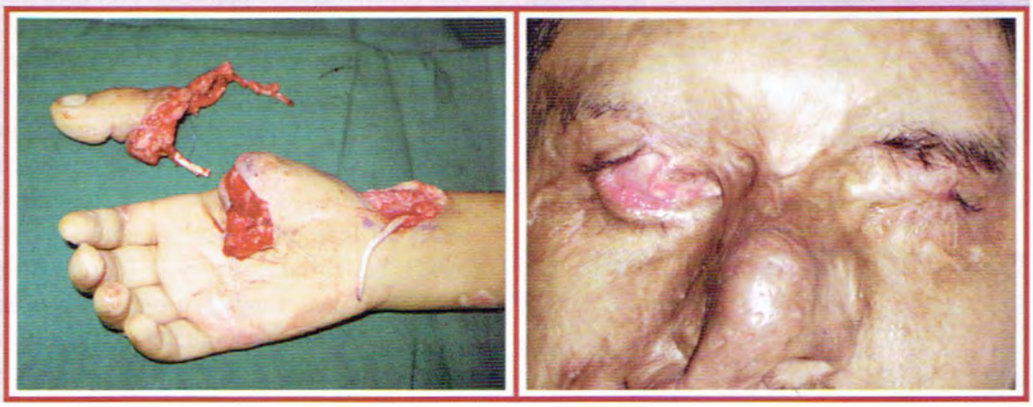
Section 5

Burns of Special Sites



Chapter 26

Facial Burns

PS Bhandari

INTRODUCTION

The face can be defined as a composite structure of bone, muscles, nerves, fat, blood vessels, skin and other tissues. However, face is more than a sum of its tissues. Rather, it is a functional, dynamic and aesthetic region. It plays an important role in formal introduction, interaction and communication. It is the physical basis of self image. Any facial disfigurement leads to great psychological trauma to the burn victim. He/she becomes crippled both socially and psychologically. Most of the scars in the body can be hidden by clothing and other means but facial scars are visible at first sight. Apart from the initial depth of burn, it is the better standards of treatment of the burned face in the acute stage that decides the improved overall aesthetic and functional outcome.

Facial burns rarely occur in isolation. Almost all the flame burns involve the face to a lesser or greater extent. Facial burns can be broadly classified into two types, one which heal within 2-3 weeks of time without or minimal scarring and those which do not heal within 2-3 weeks time (Figs 26.1 and 26.2). The latter category of facial burns lead to scarring and contraction (Fig. 26.3). This type of burn injury requires surgical intervention. Unfortunately, there is no objective measurement which can predict whether a facial burn will heal of its own or will require surgical intervention. This is because the appearance of burn wounds changes with time and also after application of various types of antimicrobial creams/ointments. Furthermore, a given anatomical depth of burn may not heal in a non-hair bearing area whereas the same depth of burn may heal reasonably well in a hair-bearing area such as bearded portion of male

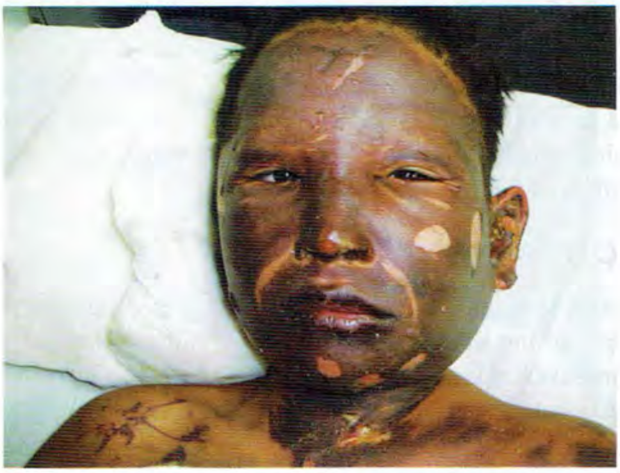


Fig. 26.1: Superficial burns of the face which are best treated by only conservative management–Exposure treatment

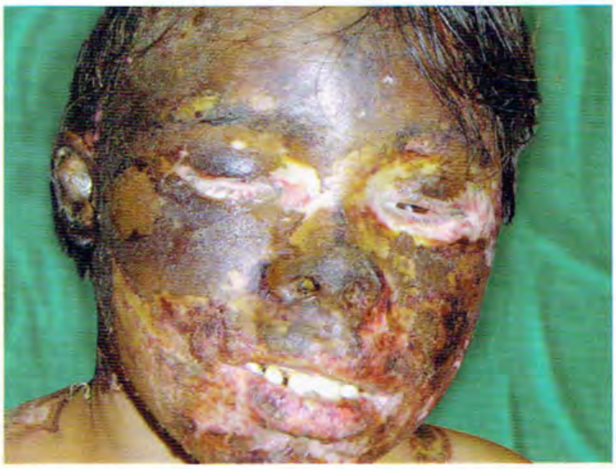


Fig. 26.2: Deep burns of the face which require surgical intervention for faster healing and to give good results

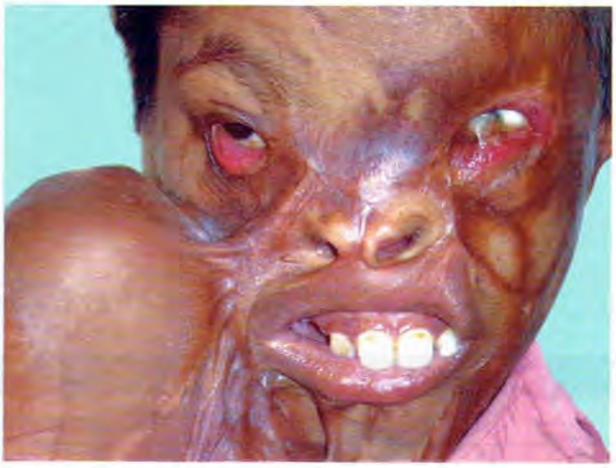


Fig. 26.3: Neglected facial burns in which wounds healed by severe scarring and contraction–Severe contraction of neck with ectropion of lids and lips and left eye corneal loss and nose deformity. These cases are very difficult to manage for correction of all the deformities (*Courtesy:* Editor)

face because of abundance of hair follicles of which majority lie in the deeper portions of dermis and even in the subcutaneous tissues. Their deeper locations make them withstand the onslaught of burn injury.

ACUTE STAGE

Management of facial burns in acute stage is a controversial topic in the overall management of burns. There is no consensus among burn specialists regarding the type of treatment, timing of surgical intervention, the technique to be used or the follow up of case. Because of its excellent blood supply, a facial burn is almost resistant to infection and has very good ability to heal. Traditionally, management of facial burn has been conservative for several reasons: (1) High potential of facial skin for spontaneous epithelialization (2) difficulty in evaluating the depth of facial burn injury (3) excessive bleeding during early tangential excision (4) Excision of subcutaneous fat may lead to severe contour deformity.

In all cases of facial burn there can be associated respiratory burn. If present, they should be given priority and be treated accordingly. Sometimes, there can be massive swelling in the neck area due to looseness of tissues. There can be disproportionate loss of fluid which may warrant intravenous fluid replacement. At times in full thickness burns around the neck, due to tough unyielding eschar, fluid can collect in the deep tissues around the neck leading to difficulty in swallowing and breathing. Patients with facial burns should be nursed in head elevated position as this reduces edema by increasing the venous return and also helps in breathing by facilitating diaphragmatic excursion.

Management: The facial burns can be managed by either exposure technique or closed technique though the former is followed most commonly. The burned area should be cleansed gently with normal saline. Mild debridement can be done but sharp debridement should be avoided as it is often difficult to determine the exact depth of injury. Flash or scalds usually result in superficial partial thickness burn which heals within 2-3 weeks of time. However, because of rich vascularity, there is large amount of oozing and soakage from the burn wound surface. This fluid forms a thick crust on drying. It can develop cracks due to dryness and may be bleed or serve as a culture medium for bacteria. An antibiotic ointment must be applied to the burn wound to prevent bacterial growth and crust formation, to soften the crust and to prevent it from cracking and bleeding until they heal. It also prevents deepening of depth of burn wound. Eyes must be protected from the topical cream applied to the face by leaving a periorbital margin uncovered by the cream or alternatively applying an ophthalmic antibiotic ointment over the face so that even if it enters into the eyes, does no harm to the eye.Topical antimicrobial creams / ointments should be replaced by wet saline or Povidone iodine impregnated gauze on 12-14 day post burn. It helps in superficial debridement of necrotic tissues. These dressings are applied wet over the wound and are removed only when dry (Fig. 26.4). Necrotic debris gets attached to the dry dressing and on removal, the necrotic tissue also comes out with the dressing. Removal of dressing in this manner is associated with some bleeding and considerable pain. Patient should be given injectable analgesics to alleviate the pain during the change of dressing. This should continue for a limited period of time till the granulation tissue starts appearing. In superficial dermal burns, the wound heal spontaneously and as the healing of the facial region is very good, the facial wounds are, therefore, best left exposed by applying local antimicrobial ointment or Vaseline gauze only. This keeps the wound lubricated, prevents its drying up and the paraffin gauze when applied and removed does not peel off the epithelializing surface (Fig. 26.5). The application of collagen dressings is also useful in such cases as it seals the wound, keeps it well humidified which encourages faster healing (Fig. 26.6). Even in chemical burns of the face the best method of treatment is to leave the wound open and dry as the wound epithelializes rapidly under the eschar and the eschar separates by itself (Figs 26.7A to D).

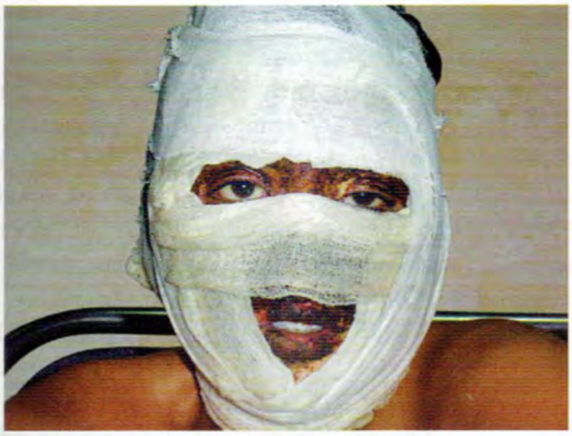


Fig. 26.4: Wet to dry dressing in case of deep burns of face for early separation of eschar

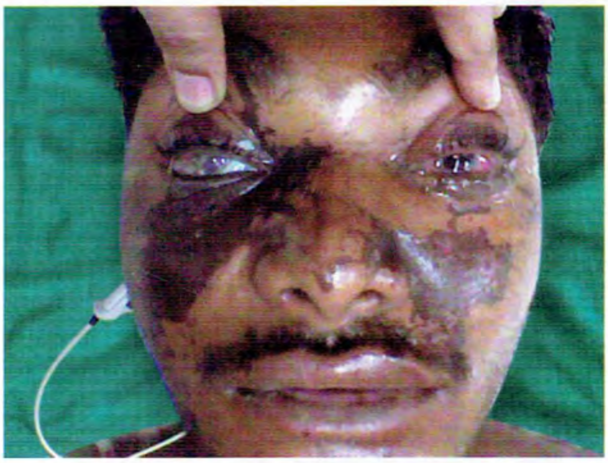


Fig. 26.7A: Superficial dermal chemical burns of face involving the eyes

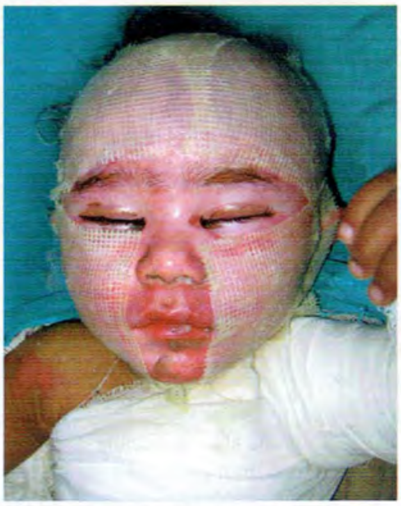


Fig. 26.5: Exposed technique of managing facial burns by applying antibiotic impregnated paraffin gauze only

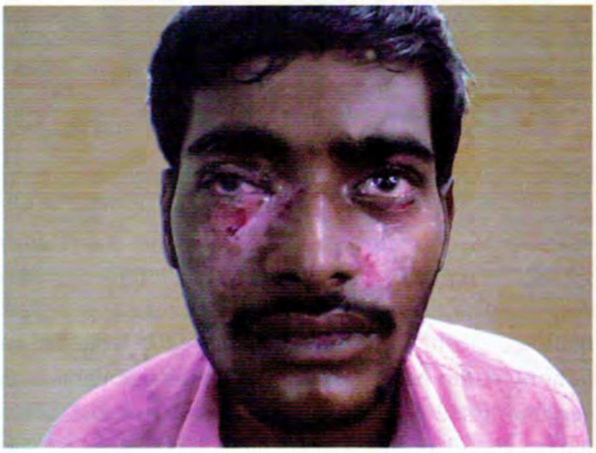


Fig. 26.7B: Ten days later almost healed wounds after leaving the wounds exposed and eschar separated by itself as the wounds healed

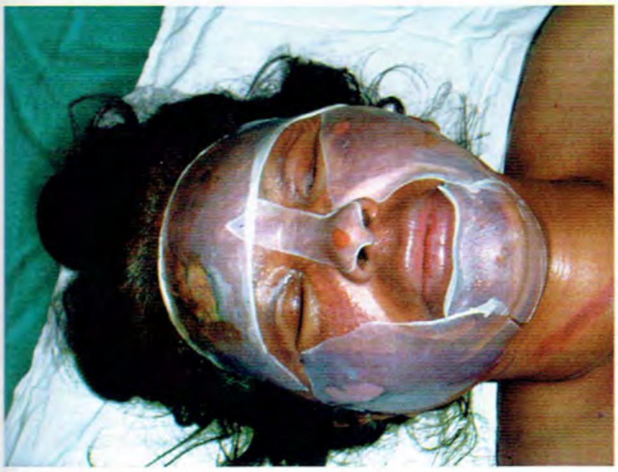


Fig. 26.6: Collagen application on facial burns

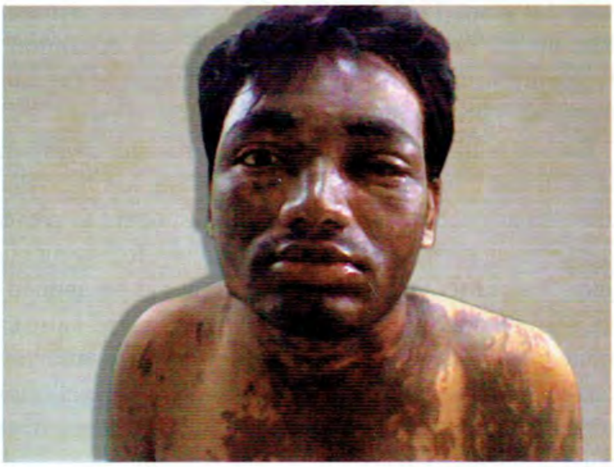


Fig. 26.7C: Another patient with extensive mixed burns due to chemical injury

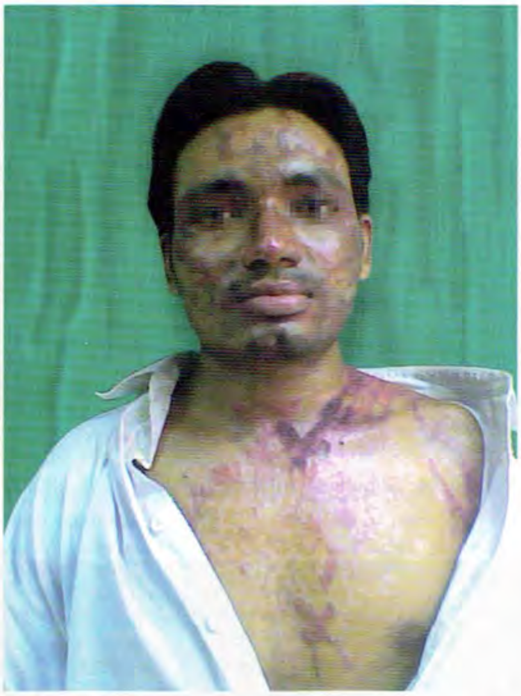


Fig. 26.7D: One month later completely healed areas after leaving the wounds exposed with only antibiotic ointment application

With the appearance of granulation tissue, use of biological dressings should be encouraged. It helps in formation of healthy granulation tissue, early wound closure and shortens the period of grafting. Surgical debridement of necrotic tissue can also be done by forceps and scissors. When the granulation tissue is healthy, it is covered by sheets of split thickness skin grafts by exposure method. Mesh graft are rarely, if ever, recommended unless the surface is bleeding profusely and there is risk of hematoma formation under the graft (Figs 26.8A and B). When the patient is taken up for grafting of raw areas, if any ectropion is developing in the lids, they can be released at the same sitting, thus, preventing any damage to the cornea.

Patients with sponteneously healed partial thickness burns and with full thickness burn where grafting has been done should be advised to avoid exposure to sunlight to prevent hyperpigmentation for at least 6-12 months following burn injury. Thereafter, sunscreen lotions should be applied if they wish to move out during the sun. Because of loose and mobile nature of tissues in the face, post burn contractures after healing and grafting are common after facial burns. Patients should be advised to do regular massage of the healed and grafted areas.They should be instructed to use pressure garment(s) 24 hours a day to prevent contracture formation and scar maturation (Figs 26.9A and B).

Although traditionally, the management of facial burn is conservative, this may sometimes lead to poor aesthetic result. Facial burns which take more than 21 days to heal run a risk of developing hypertrophic scarring. Several authors[1-5] have reported gratifying results after early tangential excision and grafting of facial burn within 3-5 days of burn injury in selected patients where the burn wound is unlikely to heal within 21 day (Figs 26.10A and B).

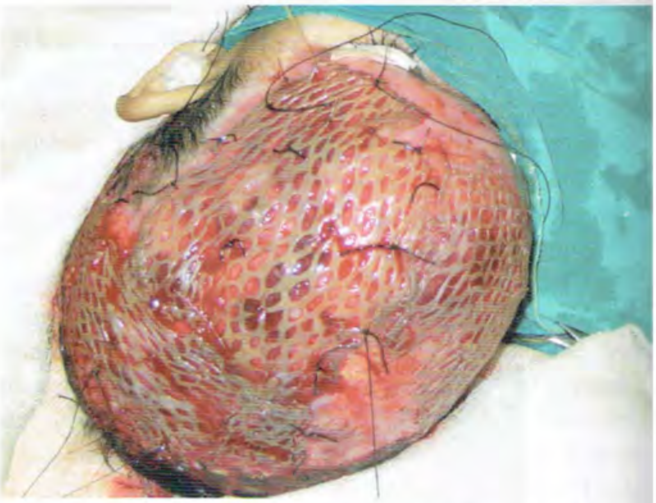


Fig. 26.8A: Post burn raw areas of scalp grafted once the granulation is healthy

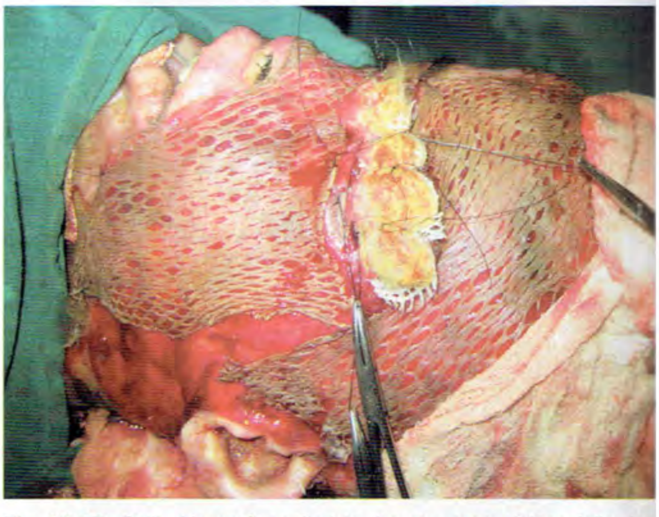


Fig. 26.8B: Split skin graft applied to the raw area of face following deep burn. Graft meshed because of excessive bleeding from the wounds. Lids also released to correct the exposed eye

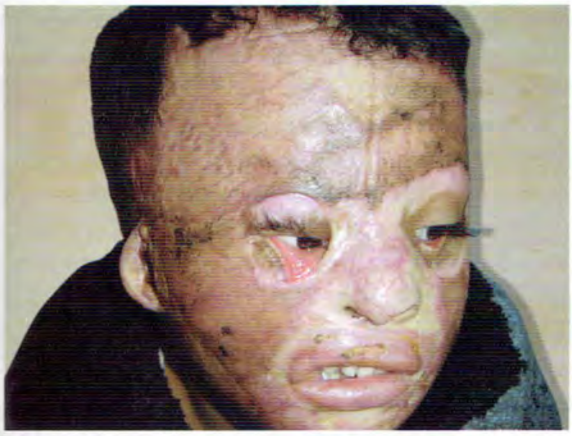


Fig. 26.9A: Postoperative picture of skin grafted post burn raw areas of face

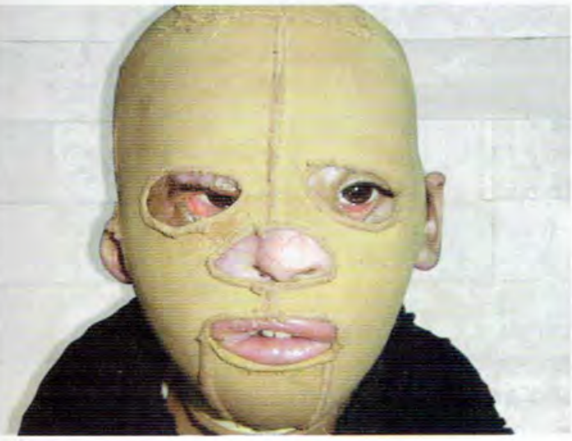


Fig. 26.9B: Pressure garment in the form of a tailor made mask to be worn over the face for preventing hypertrophy of scars

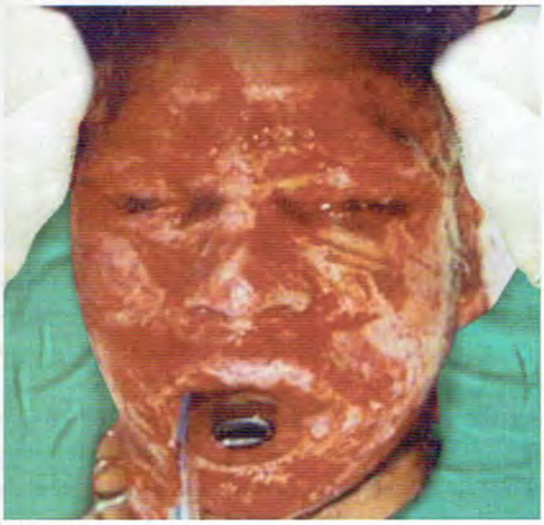


Fig. 26.10A: Preoperative picture of early tangential excision of deep burns of face

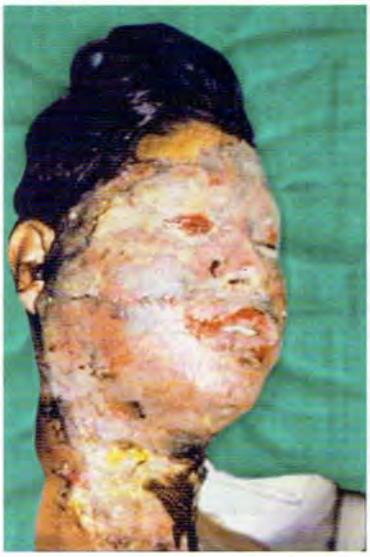


Fig. 26.10B: Postoperative picture of same patient with full 'take' of skin graft over the excised area

POST BURN FACIAL SCARRING

Severe facial disfigurement is a devastating physical handicap in which depression and social isolation are common. Facial disfigurement elicits anxiety, fear and a wish to remove it from one's sight. For scarred facial structures, thick split thickness skin graft[6], full thickness skin graft[7] and various types of flaps[8-13] have been described by different authors to improve aesthetic look of the burn patient (Figs 26.11A to C). Tissue expansion is a good option to transfer normal skin to the scarred areas. Face transplantation is also a possibility now. Face transplantation may be expected to not only improve the appearance but also to provide remedy to the prejudice and discrimination attached to this stigma. When the surgeon thinks of face transplant he must compare the risks and benefits of this innovative procedure with known risks and benefits of autogenous facial reconstruction. As on today, following face transplantation, the possibility of life threatening consequences of immunosuppression, the likelihood of graft rejection and/or graft failure appears more than the gain of receiving a normal face as compared to a surgically reconstructed face with autogenous tissues. Risks are too high when compared with the relative gain of normal facial appearance versus a reconstructed face.

MANAGEMENT OF BURNS IN DIFFERENT PARTS OF THE FACE

Nostrils: Nostril should be carefully cleaned and a thin film of lubricating antimicrobial ointment should be applied with

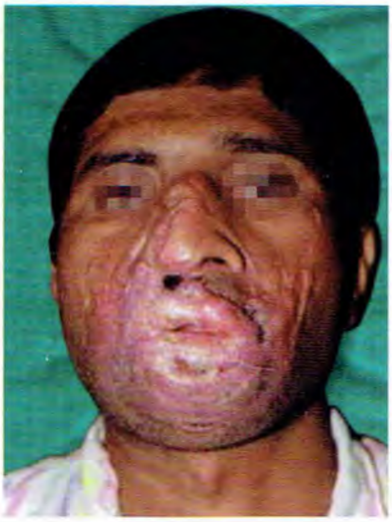


Fig. 26.11A: Post acid burn hypertrophic scarring with stenosed right nostril and severe microstomia

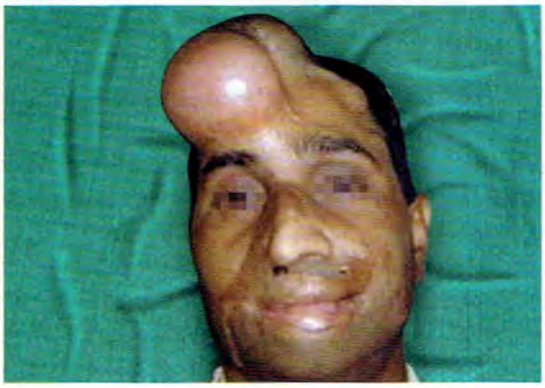


Fig. 26.11B: Release of microstomia and excision of hypertrophic scar and coverage of lower lip and chin with expanded neck flaps and nose and upper lip with skin graft. Second stage tissue expander placed in forehead and scalp for reconstructing nose and upper lip

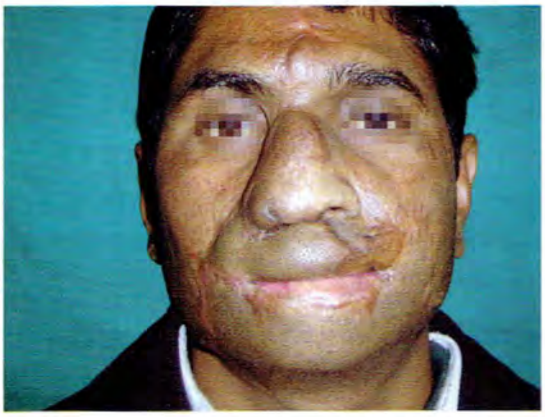


Fig. 26.11C: Reconstructed nose and upper lip with expanded forehead and scalp flap

a cotton applicator to prevent crust formation which can compromise breathing. In case of deep burns of the nose where the wounds heal by secondary intention there can be severe stenosis of the nostrils. This can be prevented by using nostril splints. In case the nostrils are stenosed due to burns, they can be opened up by release and skin grafting. In such cases, repeated release may be required because chances of re-stenosis are high and postoperative splintage is required to prevent it. This can be treated by another procedure called "Starplasty", in which two incisions perpendicular to each other are given in skin and mucosa to raise 4 skin flaps and 4 mucosal flaps (Figs 26.12A to C). The skin and mucosal flaps are then interposed with each other in the form of a 'Star' to achieve a good nostril opening. Hence, this procedure has been given the name of Starplasty. The main advantage of this procedure is that it not only gives a wide opening but the interposed flaps break the scar line, preventing the scar re-contracture. Thus, the chances of re-stenosis are minimized. No postoperative splintage is required as no graft is applied.[14]

Hypertrophic scars on dorsum of nose can be excised and resurfaced with split skin graft. If entire nose is destroyed, the forehead or arm tube flap is used to reconstruct the nose in stages, with costal cartilage for support.

Eyelid and Globe Burn

Injuries to the globe are rare in thermal injuries due to protective mechanism of blink reflex and reflex protective movement of the head at the time of injury. Nevertheless in all patients of facial burn, corneal injury must be ruled out by thorough clinical examination. Ophthalmologic examination must be done early, because subsequent periorbital edema makes the examination almost impossible, if delayed. Foreign body sensation in the eye is suggestive of corneal injury and must be ruled out or confirmed by fluorescin test. Occasionally, cloudiness of cornea is observed with flash injuries which clear out in the next few days. Corneal injuries are common in chemical burns.

All chemical burns of the eye are potentially serious.[15-18] Damage is caused by a high or a low pH and by collagenases released by damaged epithelial cells and granulocytes. This inhibits the epithelial regeneration leading to corneal ulceration. The actual damage depends upon the volume of the chemical, its concentration, its duration of contact and the unique property of some chemicals. Ammonia can penetrate to the interior of globe within 30 seconds. Alkalis

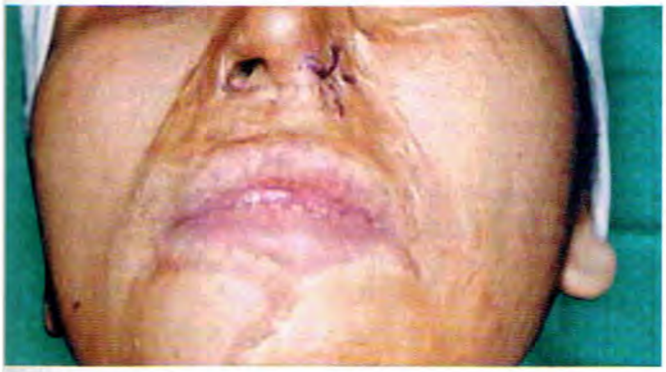


Fig. 26.12A: Post burn nasal stenosis

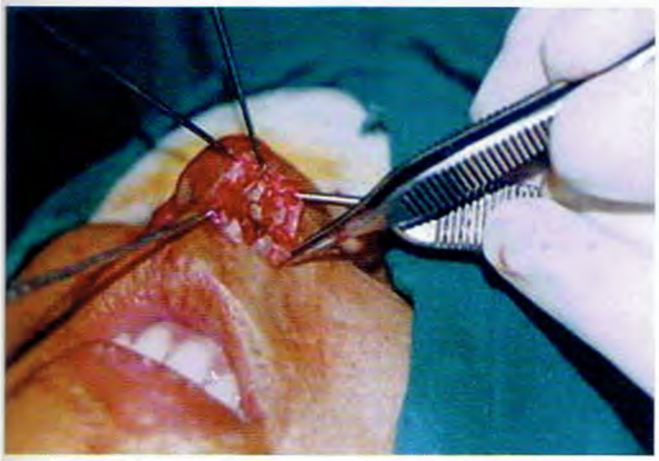


Fig. 26.12B: Starplasty—Mucosal and skin flaps raised

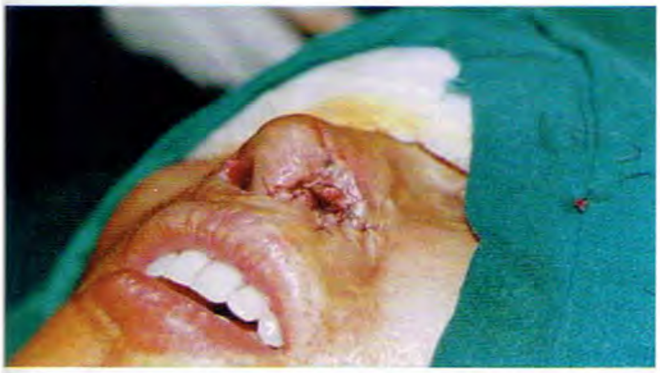


Fig. 26.12C: Interposed mucosal and skin flaps in the form of a star

are more dangerous than acids because acid produce immediate protein precipitation which, to some extent, protects the cornea whereas the OH ions rapidly penetrate the cornea producing corneal ulceration and potential for phthisis bulbi. Lime burns are notorious with a persistent pH of 11-11.5 due to particles lodging in the conjunctival sac that are not easily irrigated away. The hydroxide ions cause saponification of plasma membrane, denature collagen and cause vascular thrombosis in the vessels of the conjunctiva, episclera and anterior uvea. Burns caused by phosphorus demand careful removal of all the particles.

If the burn injury is chemical in nature, irrigation of the eye with copious amounts of normal saline is done to minimize the injury to the eye. Ideally, this should be initiated at the scene of injury with even plain water and should continue till the arrival of the patient in the emergency department. Litmus paper is useful in knowing that a normal pH has been achieved. Specific diluent for a particular agent is not required.

Subsequent care of the eye focuses on cleaning of eye with normal saline and maintenance of adequate lubrication by antibiotic drops or ointment to prevent corneal desiccations (Figs 26.7A and B). A corneal abrasion may progress to corneal ulceration and ultimately perforation with the potential loss of eye (Fig. 26.13). Cornea should be frequently evaluated. If a corneal injury is present, topical antibiotic drops and atropine should be started to prevent ciliary spasm. Chemical injuries may also require maneuvers to prevent adhesion from forming between conjunctival surfaces (Fig. 26.14A). All corneal injuries should be covered (patched). Intense periorbital edema in the early postoperative period serves the same purpose. Subsequently, if lid retraction occurs specific methods to prevent corneal exposure are necessary. Examples include scleral lens, antibiotic ointment or some other technique. Tarsorrhaphy is one of the means to close the eye.

The use of tarsorrhaphy in the acute management of eyelid burns and their subsequent reconstruction is controversial.Tarsorrhaphy involves the surgical approximation of eyelid margins (Figs 26.14C and D). Two techniques are generally advocated to achieve adherence of the eyelid margin, the opposition technique and the tongue in groove technique. In apposition technique, the epithelium of margins of both the eye lids is surgically removed and then both the eyelids are sutured with the help of bolster sutures to prevent scarring of eyelids. Tongue in groove technique ensures a stronger union between the two lid margins. However, it leads to significant distortion of lid margins.

In cases where major portion of eyelids and lid margins have been destroyed significantly, local conjunctival flaps can be used to cover and protect the eyes.

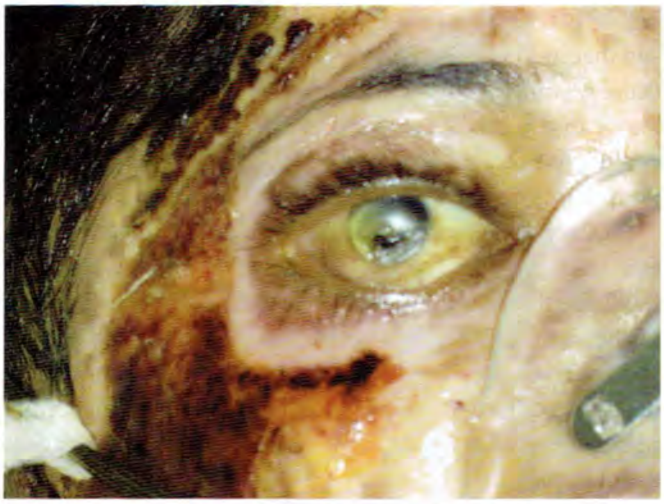


Fig. 26.13: Corneal ulceration /impending perforation

When most of the skin of both eyelids is destroyed, masquerade procedure is used to protect the eye. The conjuctival flaps are developed by dissecting conjuctiva from surrounding tissues. After suturing the flaps together with absorbable sutures, it is covered by split thickness skin graft (Fig. 26.14A).

After eyelid edema subsides and as the healing progress, the eyelids may contract, leading to exposure of globe which may cause conjunctivitis or corneal ulceration. If the lid retraction occurs with risk of corneal exposure, a temporary tarsorrhaphy can be performed to achieve the lid closure (Fig. 26.14B).

If timely debridement and grafting for deep burns of eyelids is not done, they tend to heal with contracture leading to ectropion of the lids. To prevent corneal damage from exposure, the ectropion of the lids should be released early even if the scars are not mature (Figs 26.15A to C). Ideally the upper lids should be released first and grafted with medium thickness skin graft followed by release of the lower lids and full thickness grafting of the defect. In case of severe ectropion, repeated release may be required if surrounding skin of face is also scarred and contracting (immature scars) (Fig. 26.16).

Ear Burn

All ear burns may lead to cosmetic deformity if not cared for properly. The cartilage of the ear is covered with a very thin layer of subcutaneous tissue and skin which is easily damaged or destroyed by burn. Cartilage has no intrinsic blood supply. It survives by diffusion from the surrounding soft tissues. If the cover, which is very thin, gets burned cartilage gets exposed and may slough out due to dry and infection. It leads to loss of supporting structure o ear and ultimately results in deformity of the ear (Figs 26. and 26.18A to E)

The external ear enjoys a special place in societ over the world. Women use wide range of jewellery pi to enhance its beauty. With changing trends in fashion w nowadays, even males adorn their ear with variet ornaments. Because of its limited functional significa management of a burned ear is often neglected. Managem

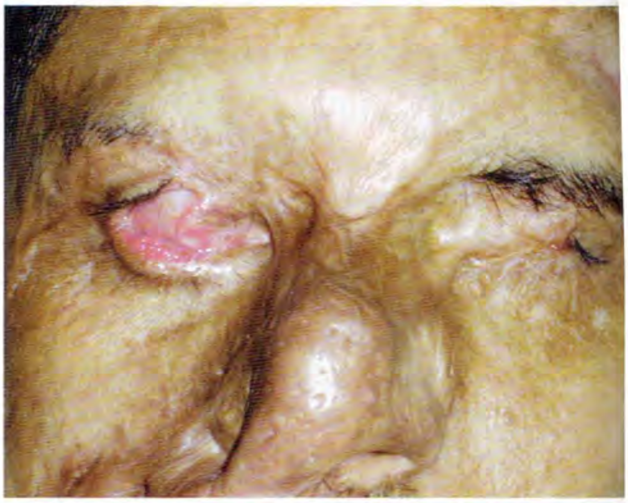


Fig. 26.14A: Symblepharosis in right eye and in left eye total los both lids for which upper and lower conjunctiva was mobilized, sutu over exposed cornea and skin graft applied over it

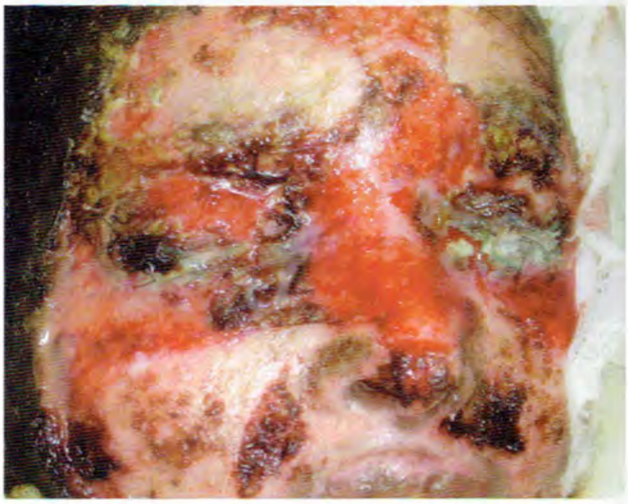
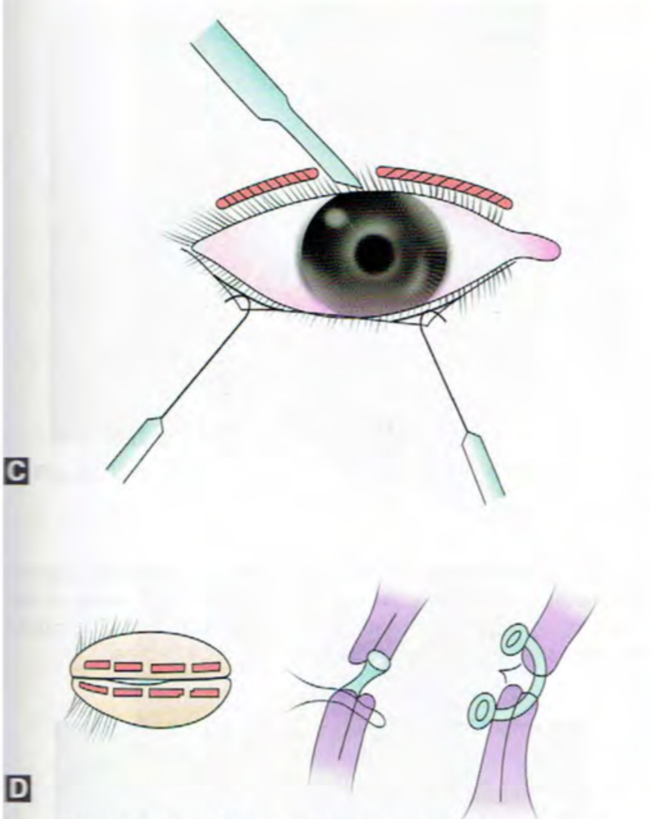


Fig. 26.14B: Deep burns of the lids with retraction of lids exposing cornea –Temporary tarsorrhaphy done in both eyes and raw a was later grafted after debriding the dead skin over the lids



Figs 26.14C and D: Line diagram to show the technique of tarsorrhaphy

pertaining to survival and function takes the precedence over burnt ear management. External ear is particularly vulnerable to thermal injury due to its prominent location on the side of the face. In most of the cases of facial burns, external ear is also involved. In burns, ear may get involved either because of direct trauma or chondritis.[19] The skin covering the anterolateral surface of cartilage is thin, adherent and unprotective and hence, is most easily damaged from thermal injury. Posterior aspect of ear is covered by thicker layer of sub-cutaneous tissue and skin which give some protection to the underlying cartilage.

In full thickness burns, the underlying cartilage gets exposed with subsequent risk of chondritis which can occur in 25% of cases. Chondritis leads to sloughing of cartilage ultimately resulting in deformity of pinna. All cases of burned ear must be treated aggressively to prevent this complication. The deformity of pinna following thermal injury can range from minor scarring to near total destruction of external ear. Because of the poor quality and quantity of skin in the auricular region, total ear reconstruction in post burn deformity is a daunting task. Every attempt should be made

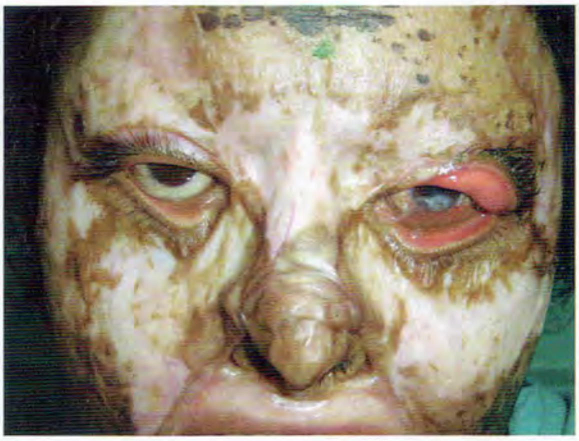


Fig. 26.15A: A case of neglected face burns developing severe ectropion of both upper and lower lids with exposure keratitis in left eye

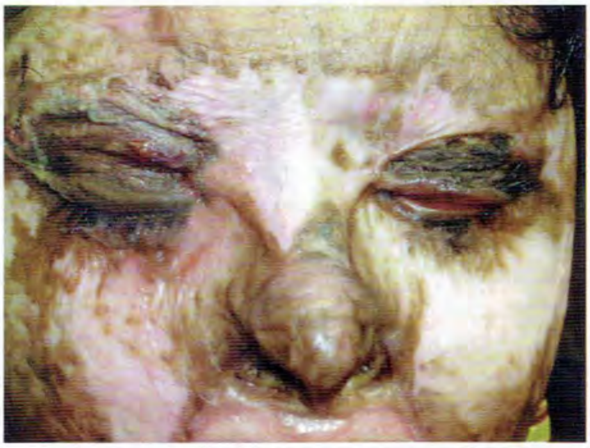


Fig. 26.15B: One week postoperative picture after release and grafting of upper lids

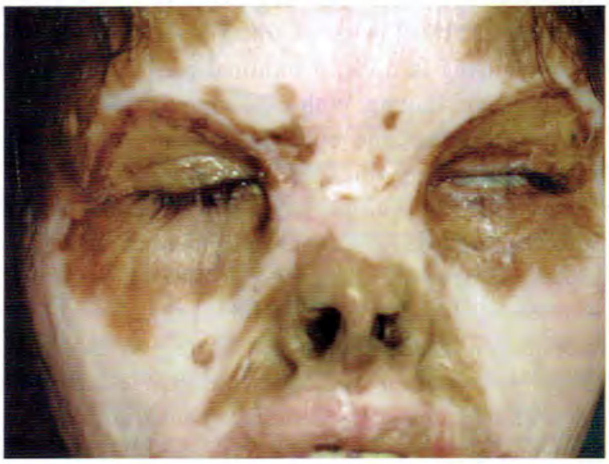


Fig. 26.15C: Late postoperative picture after release of all 4 lids with well settled grafts

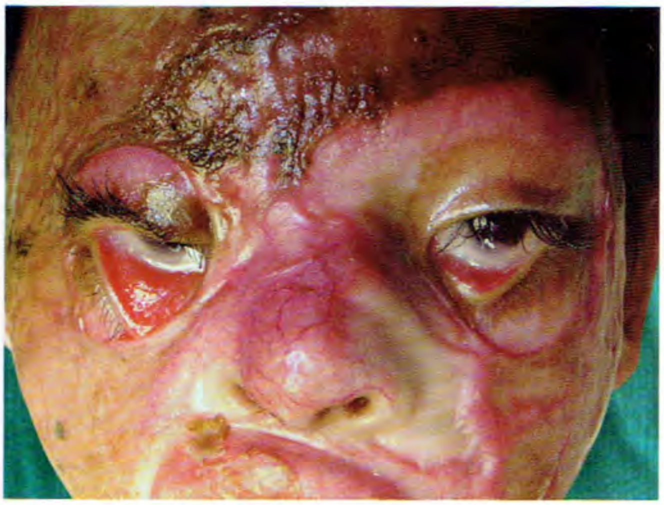


Fig. 26.16: Severe ectropion of both lower lids with immature scars in the surrounding face. Leading to persistent watering from the eyes and exposure of conjunctiva and lower cornea. Requires early release with grafting and may even need re-release because of contraction of the surrounding immature scar

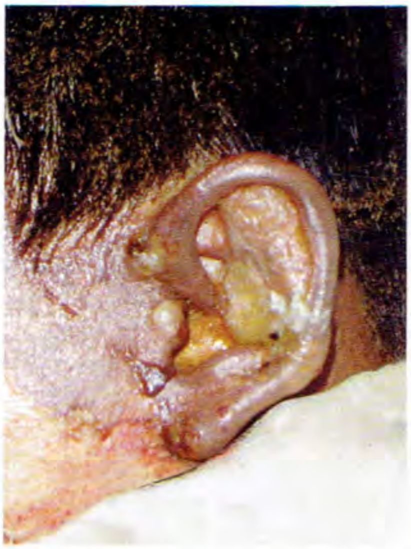


Fig. 26.17A: Acute ear burns showing how the thin skin over the anterolateral surface of the ear gets blistered and lifted off the underlying cartilage, thus impairing the blood supply to it. This can be prevented by early contoured dressing of the burned ear

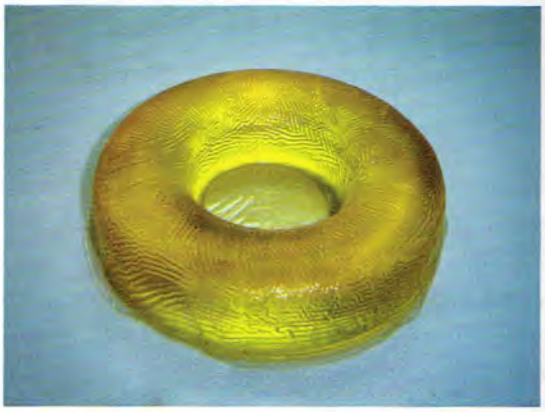


Fig. 26.17B: "Donut" used for ear burns

to prevent a deformity. Majority of ear burns heal without surgical intervention. To avoid pressure which can harm the burnt pinna, pillows are never used under the head and the patient is not allowed to lie on either side. A 'donut' can be kept under the head and it keeps the head lifted up without the ears touching it. Hence, there are no chances of pressure necrosis on the burned ears (Fig. 26.17B). Application of local antibiotic cream / ointments and avoidance of pressure help in preventing the development of chondritis in deeper ear burns. Mafenide cream is the most effective topical agent used in ear burn. In addition to topical cream, protecting the ear from external compression also helps in preventing pressure necrosis and further progress of skin loss. Usually this is achieved by the used of soft foam around the ear. If contour dressing is done, a minimal pressure should be applied while bandaging. With local wound care, application of mafenide cream and contour dressing, the swelling subsides and partial thickness burn heals with time. If perichondrium has been preserved, it will 'take' the graft. If cartilage is exposed in a small area, debridement and split thickness skin graft over the cut end of cartilage is recommended. In a large area of full thickness burn where perichondrium has been damaged leading to exposure of cartilage, some form of vascularized coverage is necessary to salvage the remaining cartilage. Unfortunately, local surrounding tissues can not be used as they are also injured and in severe burns. Regional or distant flaps[20, 21] have been described to cover the exposed cartilage. These include platysma myocutaneous flap and the temporoparietal facial flap. Temporoparietal facial flap is a thin pliable and very versatile flap and is the best choice for coverage of exposed cartilage (Figs 26.18A to E). Whether the best option should be exhausted for primary coverage of exposed cartilage or not, is a difficult decision. Besides this, weak ear cartilage may not withstand the forces of contractions of split skin graft which is applied over the temporoparietal fascia. Hence, it leads to deformity. Post auricular fascia, if not damaged by burn, can also be used to provide cover to the exposed cartilage. Rosenthal[22] has described 12 zones of the partially burned ear which are divided into three major regions which include six zones that may be used for donor

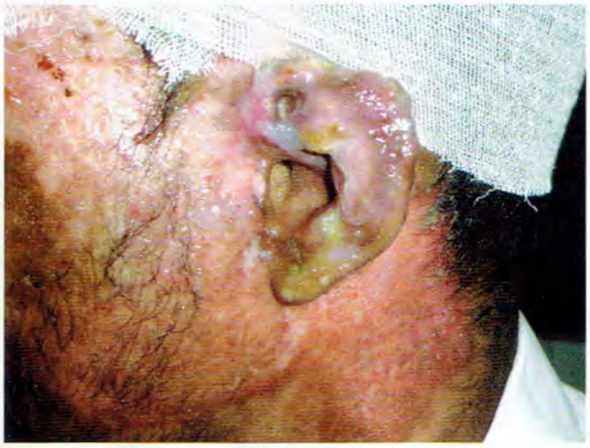


Fig. 26.18A: Deep burns of the ear with cartilage exposed after debridement of dead skin

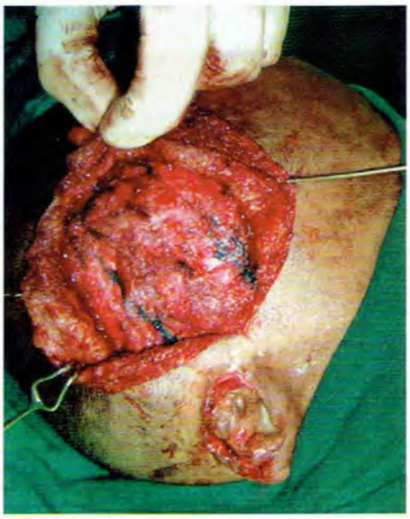


Fig. 26.18B: Temporoparietal fascial flap exposed and marked

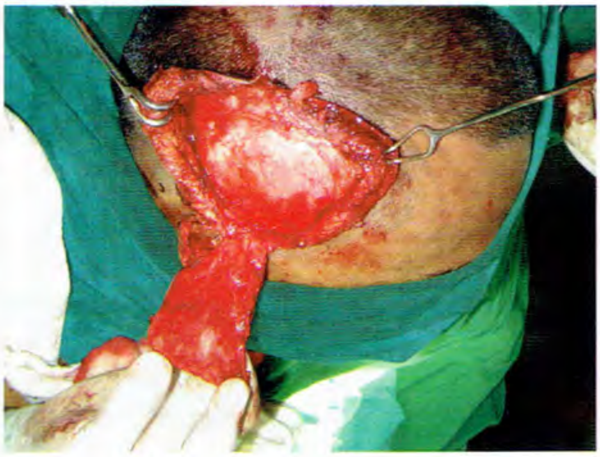


Fig. 26.18C: Raised temporoparietal fascial flap

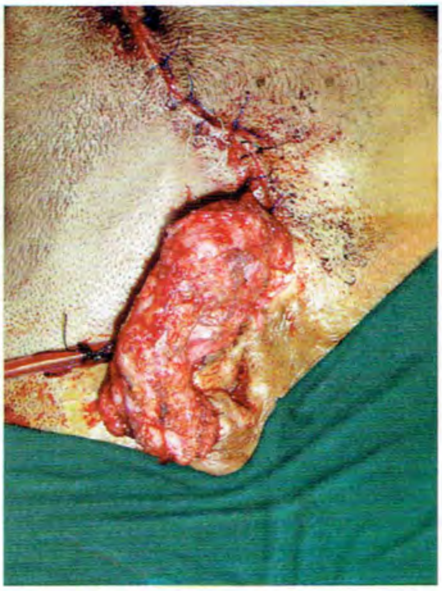


Fig. 26.18D: Flap inset over the exposed cartilage

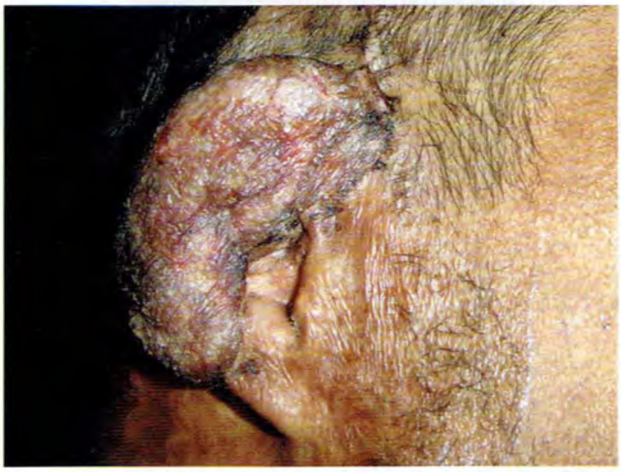


Fig. 26.18E: One week postoperative picture with full graft take over the flap – Cartilage was therefore saved

sites for advancement and resurfacing, three zones which may excised and closed without significant deformity and two zones which should be preserved, if at all possible.

Chondritis is a dreaded complication of burns of the pinna. It is characterized by rapid onset of pain, tenderness and erythematous swelling of external ear (Figs 26.19A to G). There is throbbing pain in the ear not relieved by analgesics. The cephaloconchal angle is increased which is considered diagnostic. Early diagnosis is essential to achieve the best results. Chondritis can develop even 4-5 weeks after the burn. The most common organism responsible for chondritis are *Pseudomonas aeruginosa* and

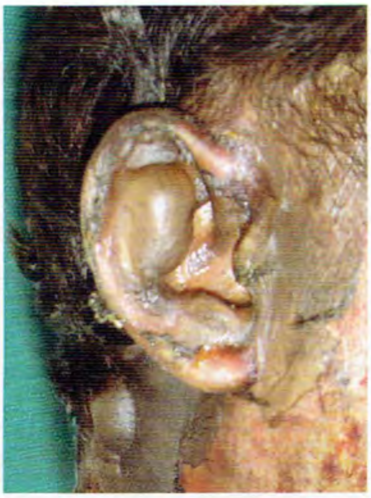


Fig. 26.19A: Superficial dermal burns of ear – Showing skin raised as a blister over the underlying cartilage

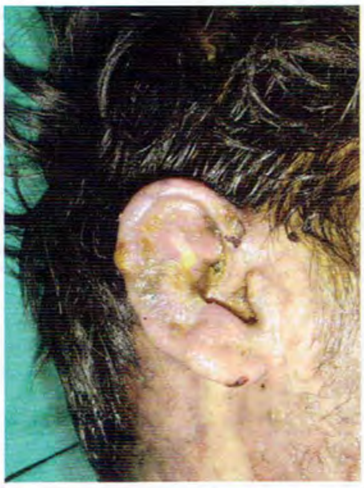


Fig. 26.19B: Same patient as in Figure 26.19A neglected and developed chondritis and chondral abscess indicated by the erythema, swelling, severe pain and tenderness

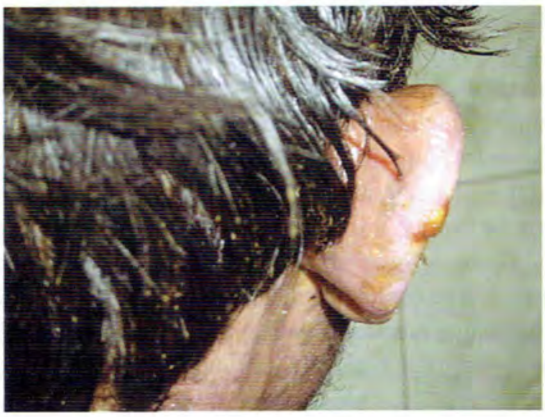


Fig. 26.19C: Increased cephaloconchal angle diagnostic of chondritis

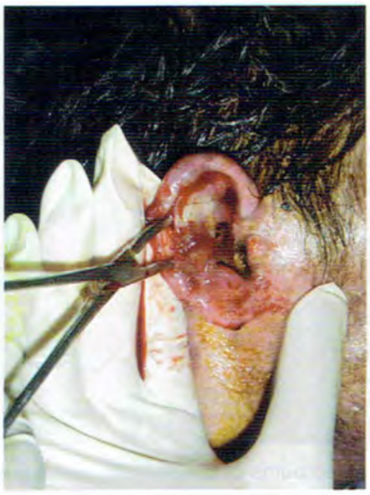


Fig. 26.19D: Incision given in the rim and pus drained and loose and dead cartilage if any removed

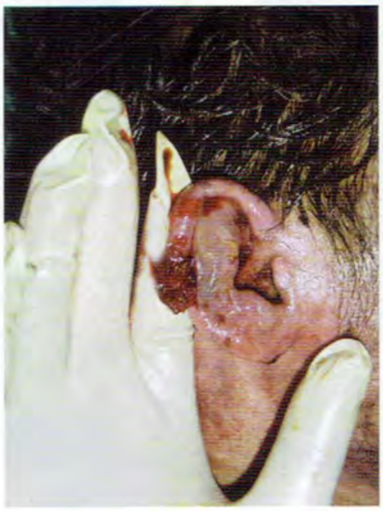


Fig. 26.19E: Cavity packed

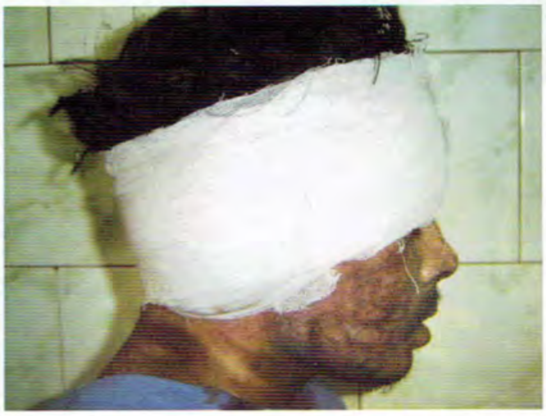


Fig. 26.19F: Contoured dressing over the treated ear

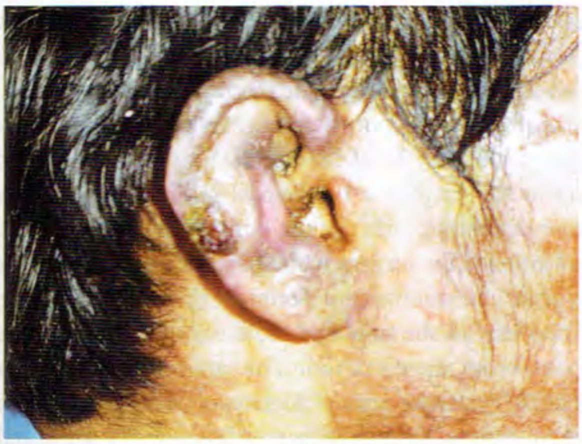


Fig. 26.19G: Same ear after I month with well maintained contours and minimal deformity

Staphylococcus aureus. The infection is treated by a bivalving type of incision. Incision is made at the helical rim that splits the ear into anterior and posterior surfaces. All infected and necrotic cartilage should be removed. The bivalved ear is packed with fine mesh gauze impregnated with antibacterial solution. A contour dressing is done with mild pressure. The dressing should be changed after 24 hours and ear should be examined for any left over necrotic cartilage which should also be excised. Dressings should continue till the wound heals.

In a post burn ear deformity, very often, the skin around the auricular region is burned and hence, not available for draping of cartilage framework. Reconstruction of ear is usually accomplished by costal cartilage framework/ Medpore® and temporoparietal fascial flap[23,24] cover or in cases where majority of the cartilage is intact but deformed, the ear can be reconstructed by using local flaps from post auricular region without adding any cartilage framework (Figs 26.20A to 26.21B).

Perioral Burns

In extensive facial burns, lips and mouth are commonly involved. In all cases of perioral burns, oral care should be given to the patients after every meal by the nursing staff and/or by the family member of the patient or by the patient himself. Oral care includes daily inspection of the mouth to see any sign and symptoms of fungal infection. If it can be spotted, treatment should be started immediately before it becomes severe enough to prevent feeding and healing of the wound. Lips are frequently burnt and antibiotic ointment should be applied to soften the crust and prevent cracking and bleeding until they heal. After every meal lubrication by

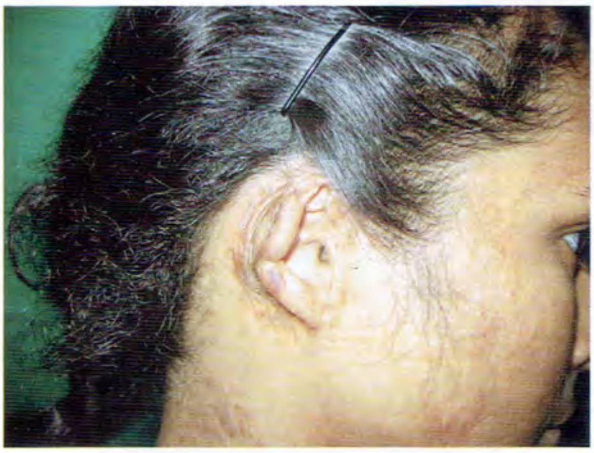


Fig. 26.20A: Post burn deformity of ear – Preoperative

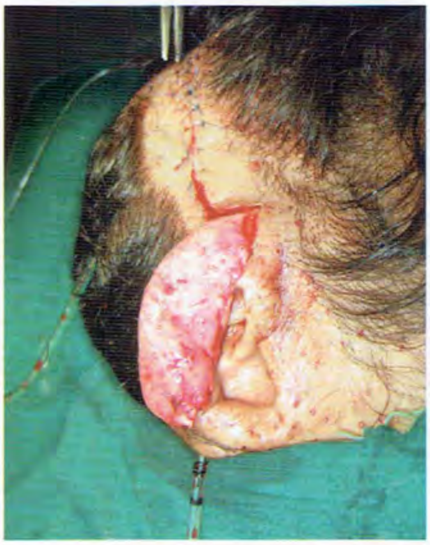


Fig. 26.20B: Costal cartilage framework draped by temporoparietal fascial flap

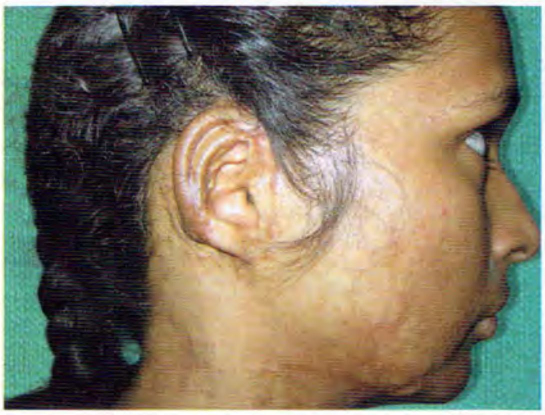


Fig. 26.20C: Postoperative result 3 months after the surgery

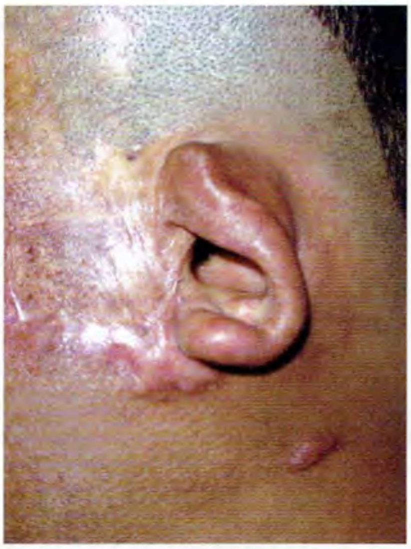


Fig. 26.21A: Post burn ear deformity with constricting effect due to loss of helical rim cartilage

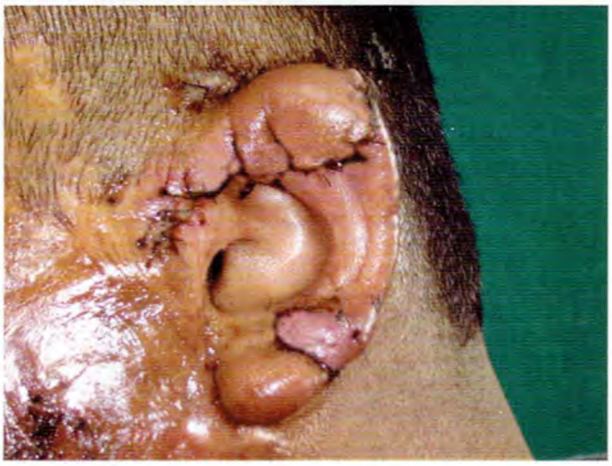


Fig. 26.21B: Ear reconstruction done after releasing the constricted ear with postauricular flaps

ointment should continue to prevent crusting. Isolated burns of lip and mouth are common in children. The majority of oral burns in children are result of a child biting on a live wire or inserting the exposed ends of an electric plug into his/her mouth. These are usually low tension injuries and the path of the current is difficult to predict because the moisture inside the mouth lowers the resistance to current flow. It may be difficult to assess the depth and extent of injury at the initial examination. It is debatable whether their primary excision should be done or else left for 10 days or so to allow clear demarcation of the zone of injury. The former may end up in excising tissues whose viability is doubtful but may eventually survive. In the latter, only definitely non-viable tissues are debrided; there is a risk of secondary hemorrhage from the labial artery in the waiting period and its possibility and the technique of control by pressing the vessel between finger and thumb should be taught to the parents of the child. All patients of perioral burn should be asked to use an oral splint during the period of scar contraction. In minor burns, the oral splints can avoid the need for surgery while in moderate to severe burns the splint can minimize deformity. However, the type of oral appliances, the time and the duration of splint therapy has not been standardized. The oral splints have been described by Hartford et al,[25] Colcleugh and Ryan.[26] Patients are advised to wear oral splints 24 hours a day for initial 3-4 months, only to be removed during eating and oral hygiene. During scar maturation patients are advised to use it for further 6 months during night only along with the massage. If proper care is not taken in acute stage in partial thickness burn and in full thickness burn, a perioral burn can result in distortion of commissure. Commissure can be reconstructed by the technique described by Converse and Wood Smith.[27] Pensler and Rosenthal[28] have recommended advancement of orbicularis muscle as a part of commissuroplasty.

Scalp Burn

Management of scalp burn is governed by depth and extent of injury. Because of its rich blood supply and high density of hair follicles, even deep partial thickness burn can heal of its own. This means that most of the scalp burn wounds will heal with conservative treatment alone. In case of full thickness burns of scalp, if underlying pericranium is intact, it will 'take' the graft (Fig. 26.22). Full thickness burns of scalp exposing the bone without pericranium require a

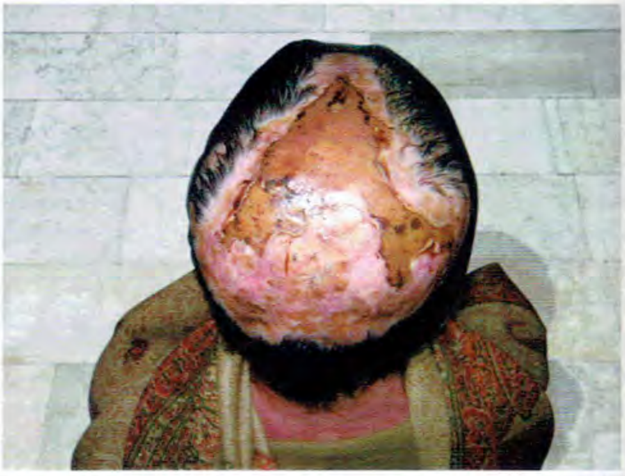


Fig. 26.22: Post burn raw area scalp covered with split skin graft

vascularized flap cover[29-31] because bare bone without pericranium does not 'take' the graft. However, the well vascularized diploe is an excellent bed for skin grafting. Two methods are available to reach to the diploic space. In one, the outer exposed and devitalized calvarial table is removed and underlying cancellous bone graft is split skin grafted. But, the graft 'take' is poor. Alternatively the outer table can be chiseled out and is covered with moist dressings or multiple burr holes, 3-4 mm apart, are made in the outer table to promote the production of granulation tissue from diploe. Granulation tissues come out through the burr hole, spread over the exposed bone ultimately meeting each other and provide a bed to accept a skin graft. Full thickness burns with exposed underlying calvarium can also be closed by rotation or transposition flap of scalp. Before mobilizing the flap, debridement of all non-viable soft tissue and devitalized bone should be done. There is a controversy regarding the amount of bony debridement in full thickness burn of scalp. Hunt et al noted a 56% complication rate associated with removal of devitalized bone. Re-growth of devitalized bone has been documented after coverage with vascularized flap. It has been suggested that limited debridement of grossly infected bone followed by immediate flap cover should be done (Figs 26.23A to D).

Eyebrow Burn

Eyebrow is a very important segment of face. It plays an important role in emotional expression of a person. Eyebrows should never be excised in acute stage even in full thickness burns or in a situation of early burn alopecia. Re-growth of

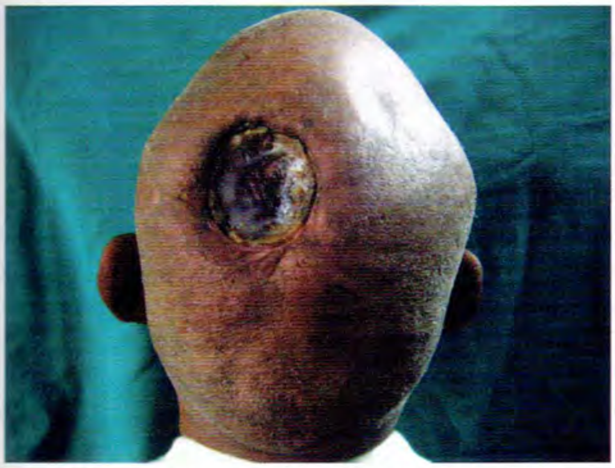


Fig. 26.23A: Post burn full thickness defect of scalp involving calvarium following electrical injury

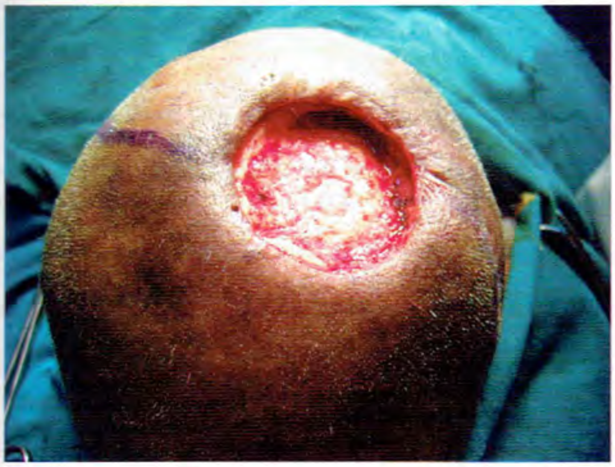


Fig. 26.23B: After debridement – Preoperative

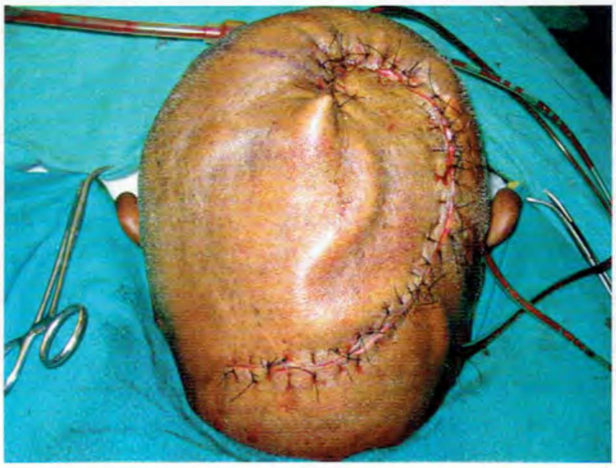


Fig. 26.23C: After coverage by rotation flap

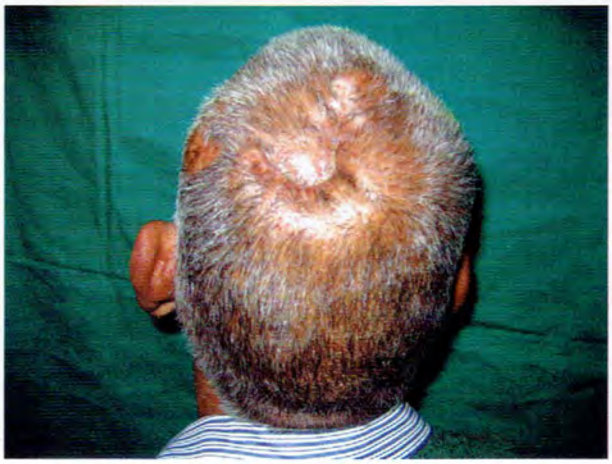


Fig. 26.23D: Postoperative result – Well healed flap

hair in eyebrow is unpredictable and can occur weeks after injury. It is because hair follicles are situated very deep in the dermis and sometimes in the sub-cutaneous tissues and hence, remain intact in deep burns. The growth of scalp hair is very long and slow, thus they appear after long period of burns. Partial hair loss within the eyebrow can be reconstructed by excision and primary closure.[32] Reconstruction of eyebrow can be done by hair transplantation, punch graft, composite strip graft, scalp flap and pedicled island temporoparietal flap based on temporal branch of superficial temporal artery[33-36] (Figs 24A to C). To restore the normal appearance of eyebrow is extremely difficult and hence, conservative management is the rule.

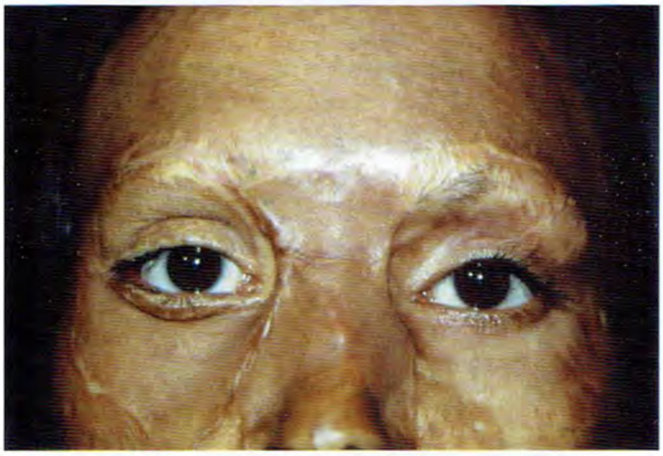


Fig. 26.24A: Post burn loss of bilateral eyebrows

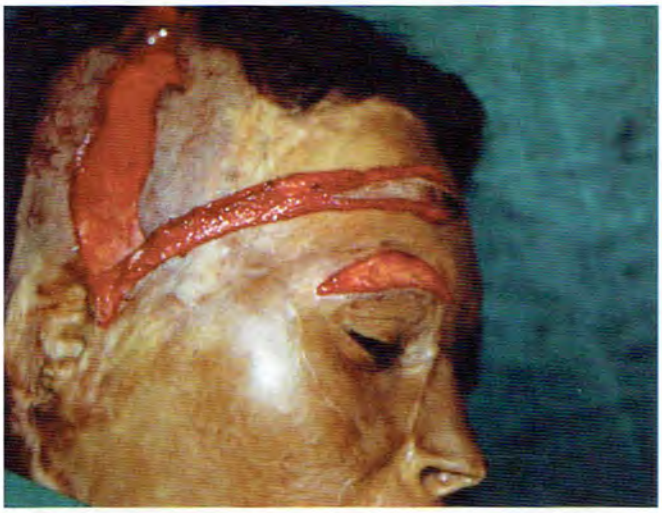


Fig. 26.24B: Hair bearing island flap based on temporal branch of superficial temporal artery

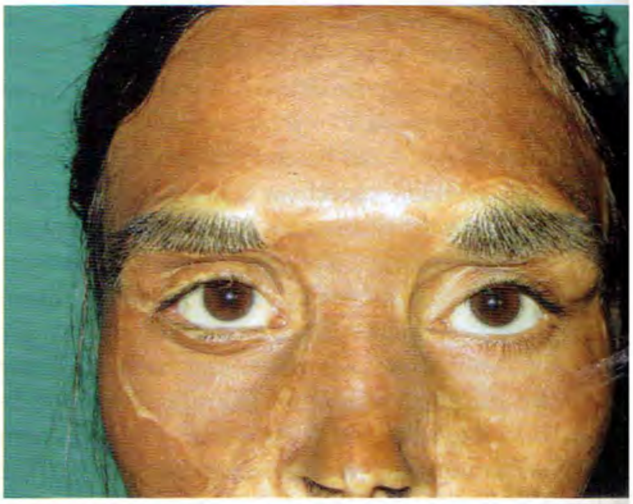


Fig. 26.24C: Postoperative result after reconstruction of both eyebrows by superficial temporal artery flap

SUMMARY

All major burns usually involve the face and isolated fac burns are uncommon. Although resuscitation of extensively burnt patient as a whole is a priority, t involvement of face cannot be ignored as it is also indicator of inhalation injury and demands immedi attention. As a number of aesthetically important structu like nose, pinna, eyebrows, etc. are located on the face, i of utmost importance to look after each of them with eq seriousness as it may affect the final outcome and hav significant bearing on the burn victim's successful ret back to society.

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Chapter 27

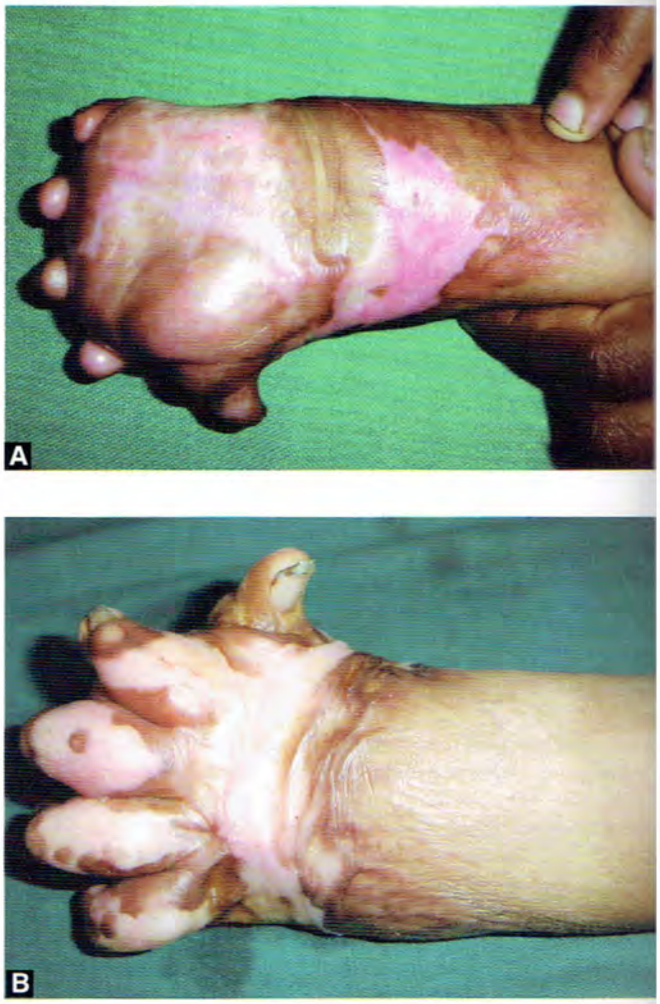
Hand Burns

S Raja Sabapathy

INTRODUCTION

Optimal hand function has a very positive impact on the quality of life of a post burn patient. This assumes considerable importance because the ultimate integration of the burn patient back into the society depends upon the functional status of the hands to carry out the daily activities and make an independent living. Tredget[1] found that in patients with a mean total body surface area (TBSA) burn of 15%, 54% of the patients sustained burns of the hand and upper extremity.

Sheridan[2] analyzed the outcome of 1047 acutely burned hands in 659 patients treated over a 10-year period. They found that in patients with superficial burns, 97% had normal hand function while in those with deep dermal and full thickness injuries requiring surgery, only 81% regained normal hand function. Although only 9% of those with injuries involving the extensor mechanism, joint capsule or bone had normal functional outcomes, 90% were able to perform activities of daily living independently. This high percentage of good function is achieved when hand burns are properly assessed at presentation and timely surgical intervention is done for those who need it. If the opportunity is missed, the result is a bad, scarred hand with severe contractures which are difficult to correct (Figs 27.1A and B). It is a preventable complication. The purpose of this chapter is to guide the surgeon to meet the specific demands of the burn injured hand and obtain the best possible outcome.



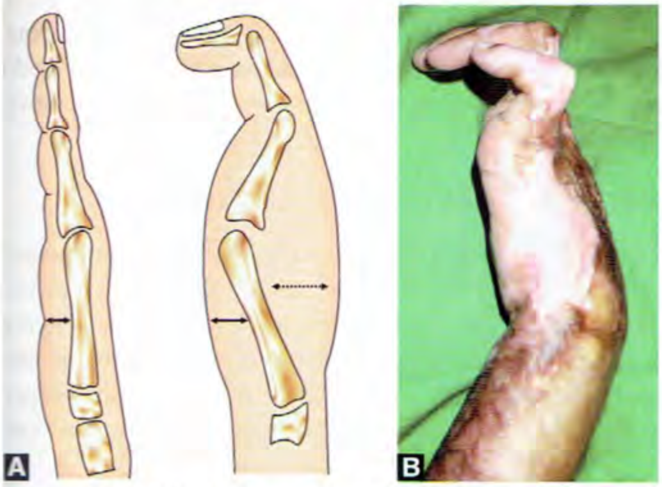
Figs 27.1A and B: An 18-month-old child who sustained flame burns to the dorsum of the hand with severe contractures due to allowing the deep burns to heal spontaneously

ACUTE HAND BURNS

Anatomical Considerations

The palm of the hand is covered with thick glabrous skin which is attached to the bone by fibrous septa. The dorsal surface is covered with thin skin, loosely attached to the underlying structures. Post burn edema causes significant swelling on the dorsum which leads to un-physiological joint positions. On the other hand, on the palmar aspect, although the edema may not be apparent, it can lead to severe physiological disturbances by causing pressure on the vessels in the palm and the median nerve in the carpal tunnel. The swelling in the palm can cause arterial insufficiency while the swelling on the dorsum can compromise the venous return. The combination results in compartment syndrome of the intrinsic muscles.

The collateral ligaments are taut when the metacarpophalangeal (MCP) joints are in full flexion and the interphalangeal (IP) joints are in extension. This is the ideal position of immobilization of these joints after injury. If stiffness occurs with the MCP joints in extension and the IP joints in flexion, the contracted collateral ligaments resist correction. The joints of hand form a system of intercalated joints wherein the distal joint takes up the deformity opposite to that of the proximal joint. The wrist joint is the key. If not splinted, the joints take up the position of maximum comfort which is exactly the opposite of what is ideal for function. In the stage of edema, if the hand is unsupported, the wrist gets flexed, the MCP joints become hyperextended and the PIP joints become flexed (Figs 27.2A and B). If the central slip gets attenuated or the dorsal skin over the PIP joint becomes thin, the stage is set for the development of the boutonnière deformity. Alternatively, if the intrinsic muscles suffer compartment syndrome, swan neck deformity develops in the fingers and first web contracture occurs. The optimum treatment of a burnt hand revolves around keeping the joints in the position of optimum function, regularly making the joints go through the full range of movement while achieving healing of the wound as early as possible.



Figs 27.2A and B: (A) The edema of the acute stage of the burns occurs predominantly on the dorsum of the hand due to the loose tissue on the dorsum. This results in hyperextension of MCP joints. (B) If the joints are not properly splinted in the acute stage, this typical deformity of wrist flexion, MCP hyperextension and IP joint flexion occurs. This is an avoidable disaster which is difficult to correct

Assessment

It is advisable to admit all cases of hand burns with the exception of very minor ones. This is because, should a complication occur, the morbidity produced far outweighs the cost and efforts involved in in-patient treatment.

If the hands are burnt as part of a major burn injury, the general management of the patient should rightfully deserve all the attention. Whatever be the total percentage of burns, a preliminary examination of the affected hand is done. One should look for associated injuries like hand and finger amputations, fractures and dislocations. The depth of burns is assessed. Second degree superficial dermal burns are pink, moist and painful and blanch on pressure. Blisters are considered to be characteristic of superficial burns. Deep dermal burns are waxy white in appearance. Full thickness burns are obvious by the thick leathery skin, appearance of thrombosed subcutaneous veins through the skin and absence of pain to pin prick. Assessing ends of extremes is easy but assessing deeper partial thickness burns can be difficult. Newer technologies like laser Doppler flowmeter, intravenous Fluorescein and ultrasound have been tried but traditional clinical assessment remains unsurpassed by new technology.[3] If the area does not blanch on pressure, is dull to pin prick then probably it is a deep partial thickness injury. The areas in question must be marked in a drawing and attached to the case records. This must be done before the application of topical antimicrobials, as otherwise the assessment may become difficult later.

In circumferential full thickness burns and deep partial thickness burns the edema can cause circulatory compromise. Ultrasound recordings, pulse oximetry, photoelectric plethysmography have been tried but escharotomy must be performed on clinical grounds. Significant increase in survival of fingers has been found following lateral digital escharotomy compared with those not decompressed.[4] If the hand starts feeling cold and palmar arch pulse disappears on Doppler, it is better to decompress the part. Escharotomy has to start proximally in the arm depending upon the involvement. If proximal escharotomy does not make the fingers warm it has to be continued to the hand and digits. In the digits it is done on the ulnar side of the index, middle, ring and little fingers and the incision can be carried proximally into the dorsum of the hand. For the thumb, the initial incision is on the radial side. Escharotomy is done with fine cautery and some patients may need anesthesia. Hemostasis must be carefully secured after release.

The edema may mask the onset of compartment syndrome of the intrinsic muscles. High levels of suspicion are necessary. The Finochietto intrinsic tightness test must be done (Fig. 27.3). The MCP joint is passively extended and when in that position the PIP joint is passively flexed. Presence of good resistance is an indication for the release of the intrinsic muscles. This requires anesthesia. The escharotomy incisions can be used to release the intrinsic compartments and usually in such instances the carpal tunnel and the Guyon's canal are also decompressed. If nerve compression is the predominant problem, claw hand deformity results (Figs 27.4A to J). If intrinsic muscle contracture is the main issue, swan neck deformity results.

Initial Management

Once assessment of the depth of injury is made and recorded and the need for decompression is taken care of, the hand is dressed with moist non adherent dressings. Dry dressings and exposure method is not recommended for hand burns. The hand is dressed with topical antimicrobials like silver sulfadiazine 1% cream. After dressing, the hand is immobilized with a splint with the wrist in mild dorsiflexion, the MCP joints in about 70 degrees of flexion and the IP joints in extension. Between two dressings, the hand should be put through possible range of movement at least twice a day. In the initial stage the dressings may need to be done daily and the movements must be started on Day 1, even in critically ill patients.

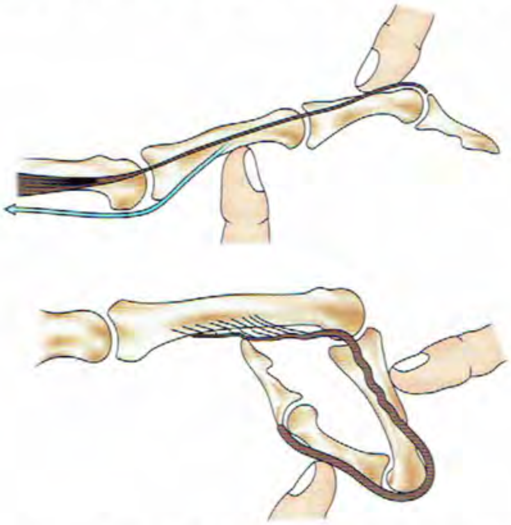


Fig. 27.3: Intrinsic tightness test (Finochietto-Bunnell). When intrinsics are tight it becomes difficult to flex the PIP joint with the MCP joint hyperextended. If this is positive then the intrinsics have to be released. When the MP joint is in extension, the contracted interosseous muscle impede flexion of the PIP joint because of the traction exerted on the extensors. Flexion of the MP joint relaxes the extensors and flexion becomes possible at the PIP joint

If the depth of burns is assessed as superficial the wound can be dressed, splinted and left as such for 4-5 days. An earlier dressing change may be indicated in case there is soakage of dressing or the patient complains of undue pain, fever or foul smell/discharge, etc. Very frequent dressing changes in such cases disturbs the healing epithelium and may increase the chances of infection. Another method suggested for the management of superficial burns is to smear the hand with silver sulfadiazine and place the hand inside a sterile surgical glove and allow continuous movement. The glove can be replaced when it gets soiled. Good healing with absence of contracture has been shown by this technique.[5]

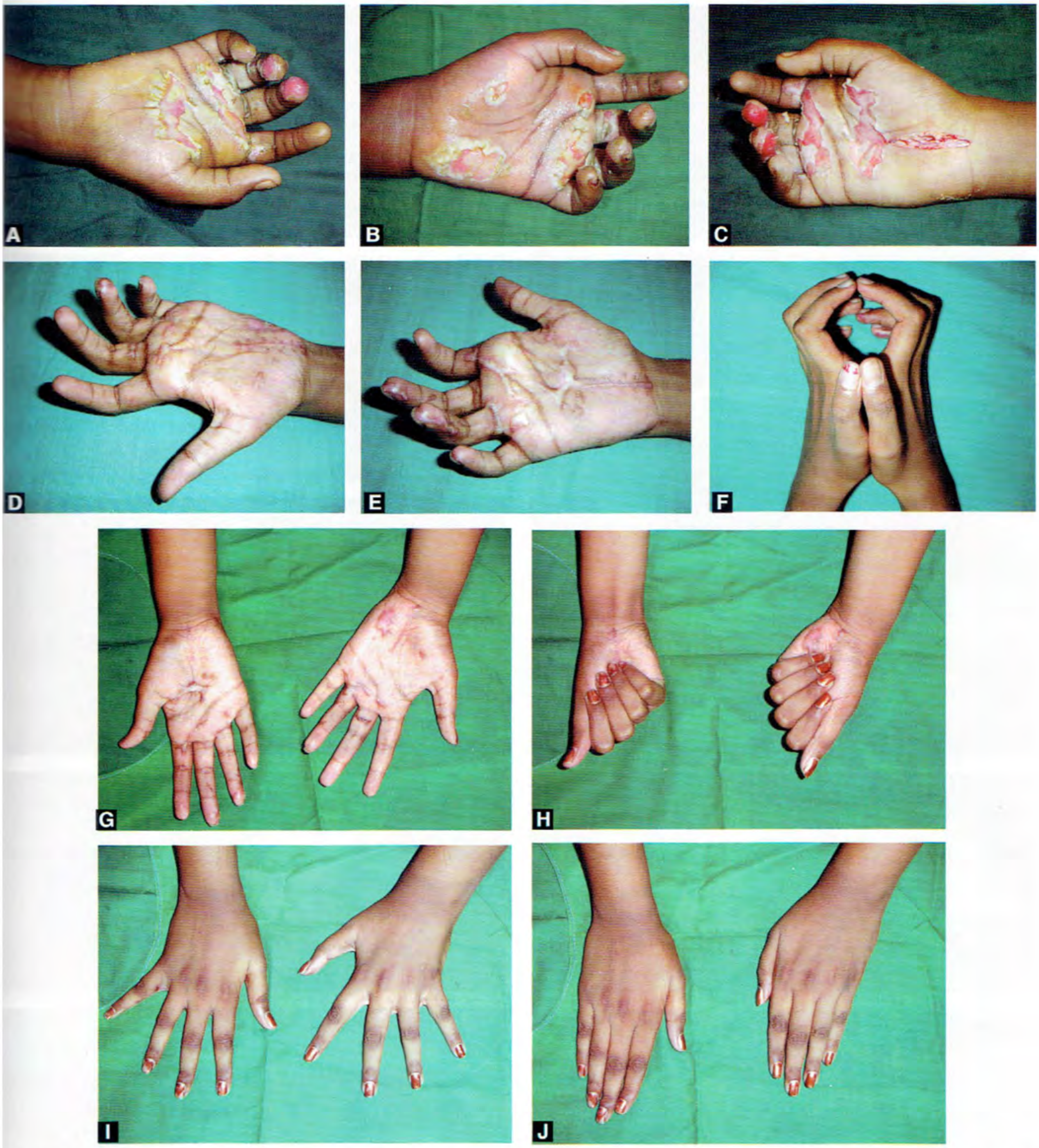
If one is unsure of the depth of burns or if the dressings get extensively soaked, the wound is redressed. Lack of adherence of the dressings to the wound is considered another indication of deep burns.[6] At all times, the hand is diligently splinted in functional position and early motion exercises started. The hand is kept elevated and in a splint at all times except when the patient is on therapy. By about 3 to 5 days the wound can be reassessed and decision made for continuation of conservative management or operative intervention.

EARLY SURGERY FOR BURNS OF HAND

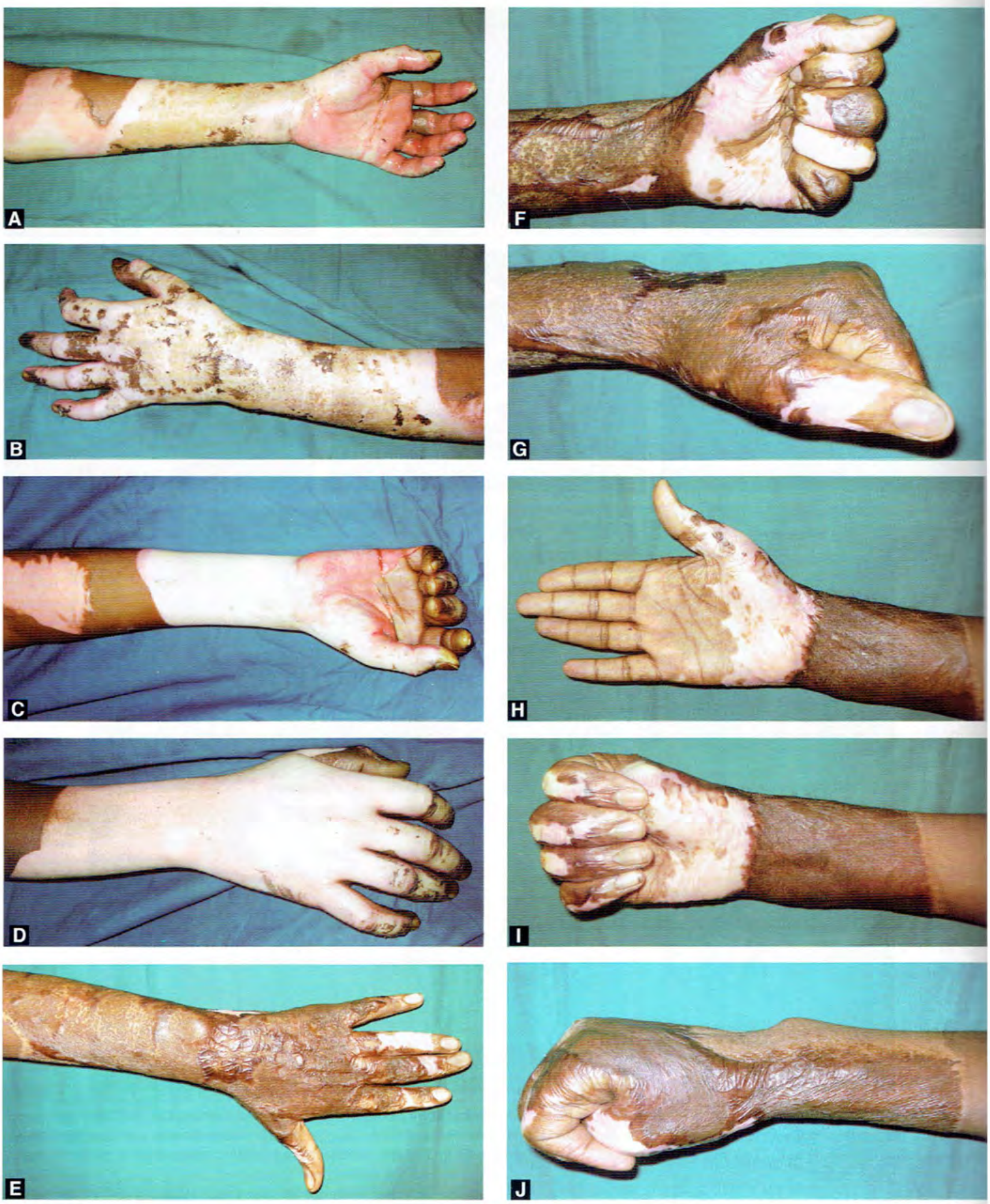
Early surgery for a burnt hand can be done for second degree deep dermal, third degree full thickness or in the fourth degree burns when underlying tendons, bone or joints are also involved. In second degree deep dermal burns, the aim is to obtain near normal function, while in the deeper degree burns the aim is to maximize the restoration of function.

Tangential Excision

Early tangential excision and coverage of burns initiated by Janzekovic (1970)[7] is a milestone in burn care (Figs 27.5A to J). It involves shaving off of the deep dermal burns up to viable dermis and immediate coverage with skin. It is done



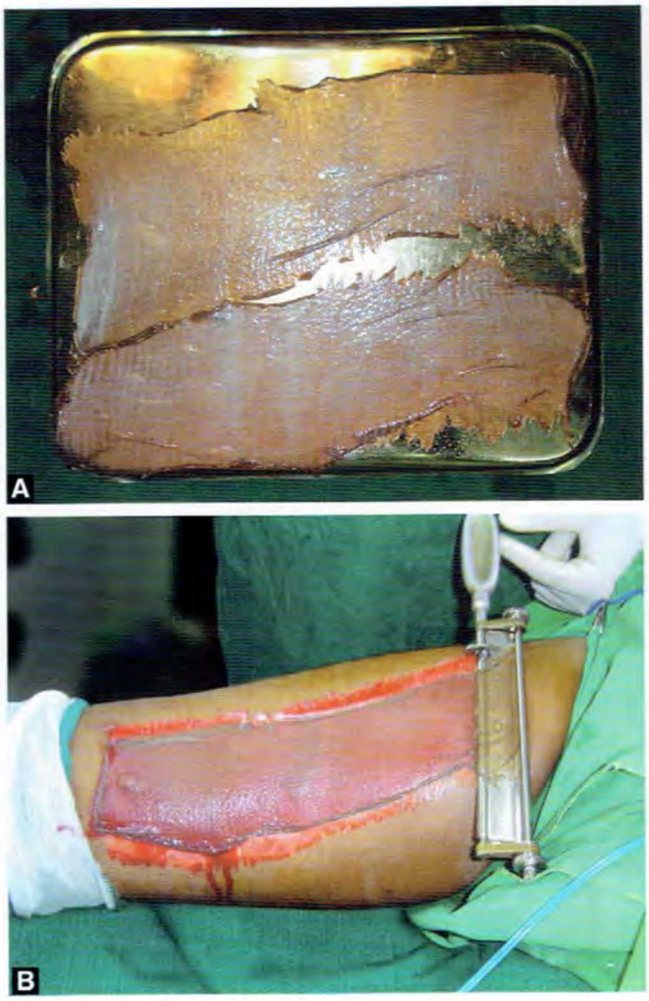
Figs 27.4A to J: A 12-year-girl with electrical burns of both hands admitted after 14 hours of injury. She had numbness in all the finger tips (A and B). Carpal tunnel and Guyon's canal decompression was done in both the hands (C). Despite this, he patient developed complete intrinsic paralysis in both hands (D to F). The intrinsics recovered in 3 months time (G to J). If the nerves are not decompressed in time, patients usually end up with gross deficit



Figs 27.5A to J: Deep dermal flame burns involving both hands (A to D). Patient had tangential excision of the burns and skin grafting. Good functional results seen during follow-up (E to J)

around 4 days post burn when the patient has recovered from the burn shock phase. The burnt surface is successively shaved with a grafting knife, set to cut a thin slice, until fine punctuate bleeding is encountered as the tips of the vertical dermal capillaries are shaved off. This is done under tourniquet and confirmed after deflation. Between the sites of dermal bleeding, dermal collagen is exposed and some epithelium remains viable in the deep skin adnexae. This surface supports a skin graft applied immediately so that it does not dry out. Grafts survive by picking up a blood supply from the capillary loops which have been transected. The intervening dermis becomes incorporated beneath the graft. Healing is faster and better cosmetic and functional results are achieved because the deeper dermis of the native skin is saved. To prevent the problem of hematoma formation beneath the graft, tourniquet is released after shaving and all brisk bleeding points are coagulated till it is felt that the fine ooze can be controlled with pressure. When all brisk bleeding points have been coagulated, the tourniquet is inflated again, grafts applied, gentle compression dressing given and then the tourniquet can be released. If there is doubt about hemostasis, the first dressing can be done at 48 hours to drain the sub graft collection.

It is ideal to apply a split thickness sheet graft with a thin layer of dermis (0.010 to 0.012 inches thickness) to cover the raw areas (Figs 27.6A and B). 1:1.5 meshing of the skin graft can be done, but it should not be expanded. In tangential excision, thick grafts and meshed grafts must not be applied. They give a poor cosmetic result.[8] Remnants of epithelium which grow after graft 'take' may produce small inclusion cysts on the surface for some months. As the cysts appear beneath the thin skin graft they can be decompressed and they settle well. In the tangential excision technique, the area shaved must always be covered with graft either immediately or at least within 2 hours. If left uncovered, the exposed dermis desiccates and the deep dermal burns get converted into a full thickness injury. The technique involves blood loss and hence should always be done under tourniquet. Blood loss is not a problem when surgery for hand burns is done under tourniquet. At any particular session not more than 10% of body surface area must be shaved and grafted. Tangential excision requires correct choice of patient, blood, infrastructure and skill. It is suitable for experienced personal. For others, the safer option is the conservative management with dressings and grafting the raw areas at the earliest.

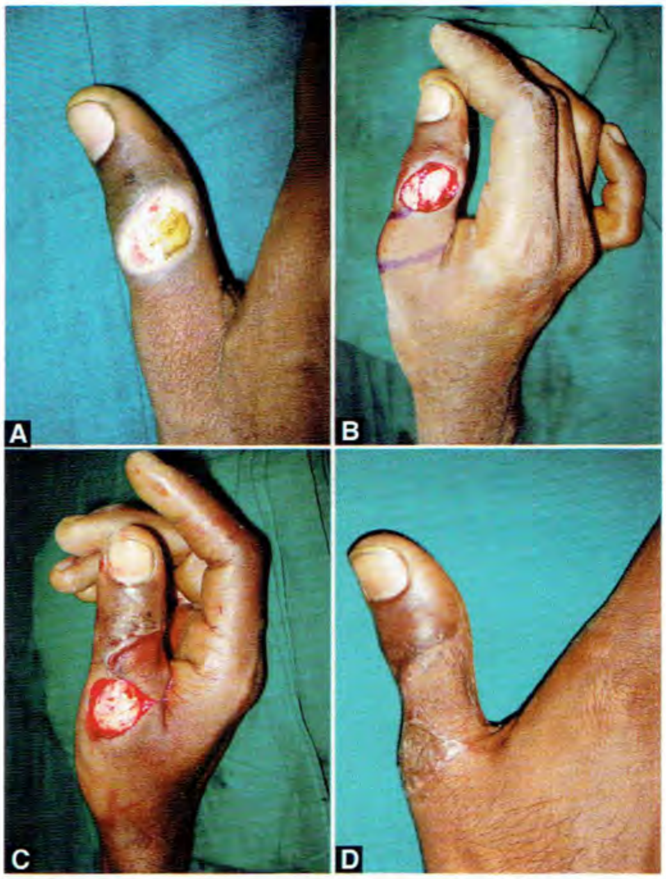


Figs 27.6A and B: When early tangential excision of burns is done the raw area must be immediately covered with thin graft. Figures show the recommended thickness of the graft

In tangential excision, a layer of viable dermis must be left behind to be covered with thin skin graft. It must not be confused with excision of the burns done for full thickness injuries which is dealt with later. Tangential excision is good for deep dermal burns involving the dorsum of the hand. It is generally not used for the palm.

Excision of Burns

When the injured area is limited, definitely full thickness and the general condition of the patient permits, the burn wound can be excised and skin cover provided. In hand burns it is usually applicable to limited area full thickness burns overlying joints, tendons and bone (Figs 27.7A to D). In such circumstances if allowed to take the natural course, the necrosing burnt area will be complicated by infection



Figs 27.7A to D: Localized deep burns of the hand can be excised primarily and covered with local flaps

and loss of critical tissue. Early excision and flap cover in such circumstances helps to salvage critical structures. In burns of upper limb, it is better to limit this excision to such instances. When done for wide areas of the forearm and arm, the level of excision has to be up to the deep fascia. Mere excision of the full thickness skin will expose the subcutaneous fat which may not be a good bed for the survival of the split skin graft.

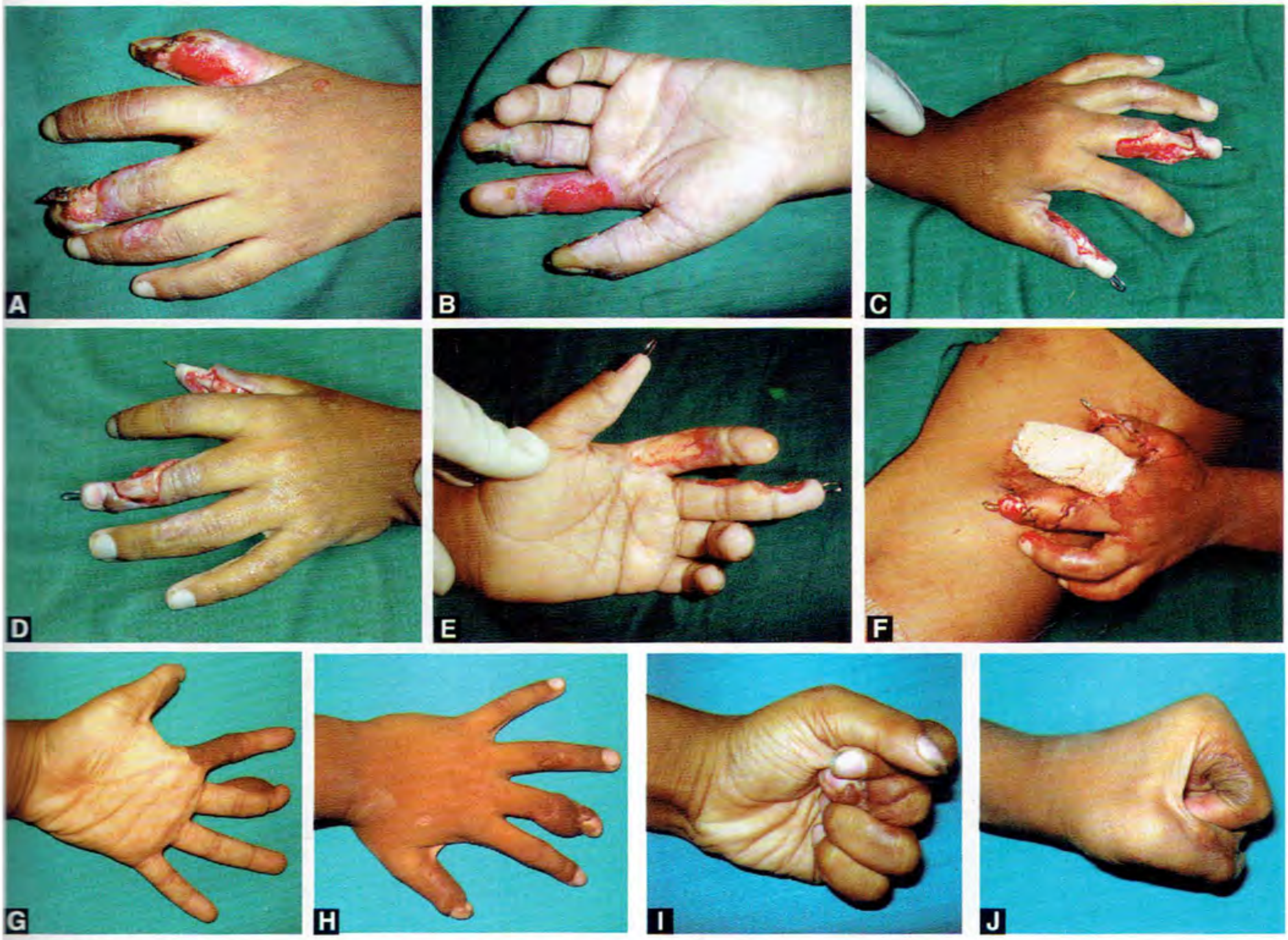
Timing of Surgery

To obtain a better understanding of the parameters involved in the long-term functional outcome of hand burns a retrospective study was performed by VanZuijlen et al on 88 consecutive patients with hand burns (143 hands).[9] Patients were followed for 12 months. Hand function was assessed by seven objective test criteria (7-OTC) described by Jebsen[10] (Table 27.1). Five parameters were found to have a significant predictive value for long-term hand function. In order of predictive value, these were finger amputations, age at admission, impaired autograft 'take', the full thickness hand burn surface area and the full thickness total body burn surface area. No relationship was found between the post burn day of operation and the long-term hand function. This finding is inconsistent with the consensus that functional outcome is improved by early excision and grafting. While provision of early cover to the hand burns is beneficial, in a patient with major burns general measures and coverage of large segment raw area takes priority. It is the opinion of this author that hand function is well preserved when burns of uncertain depth are treated expectantly, followed by selective debridement and grafting. Advantages include reduced blood loss, no loss of vital tissue and a reduction of the need for donor sites.

If a diagnosis of full thickness burn of the hand is certain, early primary excision and grafting is advocated. If diagnosis is a clear deep dermal second degree burns of the dorsum of the hand and fingers, tangential excision and grafting is done within 4-5 days. Rehabilitation is commenced as early as possible and the length of hospital stay is reduced. If the assessment of burn depth is difficult, the operation can be postponed to prevent excision of vital tissues allowing those areas with partial thickness burns to heal spontaneously. A 'wait and see' approach to hand burns of uncertain depth is valid. The function of the hand will not be adversely affected by delayed surgical treatment in those patients with extensive burns in whom early large area wound closure takes priority.[9] The aim is to achieve skin cover to the hand within 2-3 weeks from injury before infection sets in. However, till wound healing is achieved the hand has to be splinted in the functional position and range of movement exercises have to be provided. After grafting, if there are areas of graft losses, re-grafting of the areas is important. Allowing healing by secondary intention results in poor outcome.

Table 27.1: Jebsen's[10] objective test criteria

1. Writing words as quickly and clearly as possible with dominant and non-dominant hands.
2. Turning over five 3 x 5 inch index cards (simulated page turning)
3. Picking up small common objects: Asked to pick up a coin and paperclip, placed on a desk.
4. Simulated feeding: Had to pick up peas from a plate with a teaspoon and bring it to his/her mouth.
5. Stacking checkers: A stack had to be made with five small wooden checkers, placed in front of the patient.
6. Picking up large light objects: The patient was asked to pick up an empty can with a diameter of 10 cm
7. Picking up large heavy objects: Was asked to pick up a full can (600 grams) with a diameter of 10 cm.



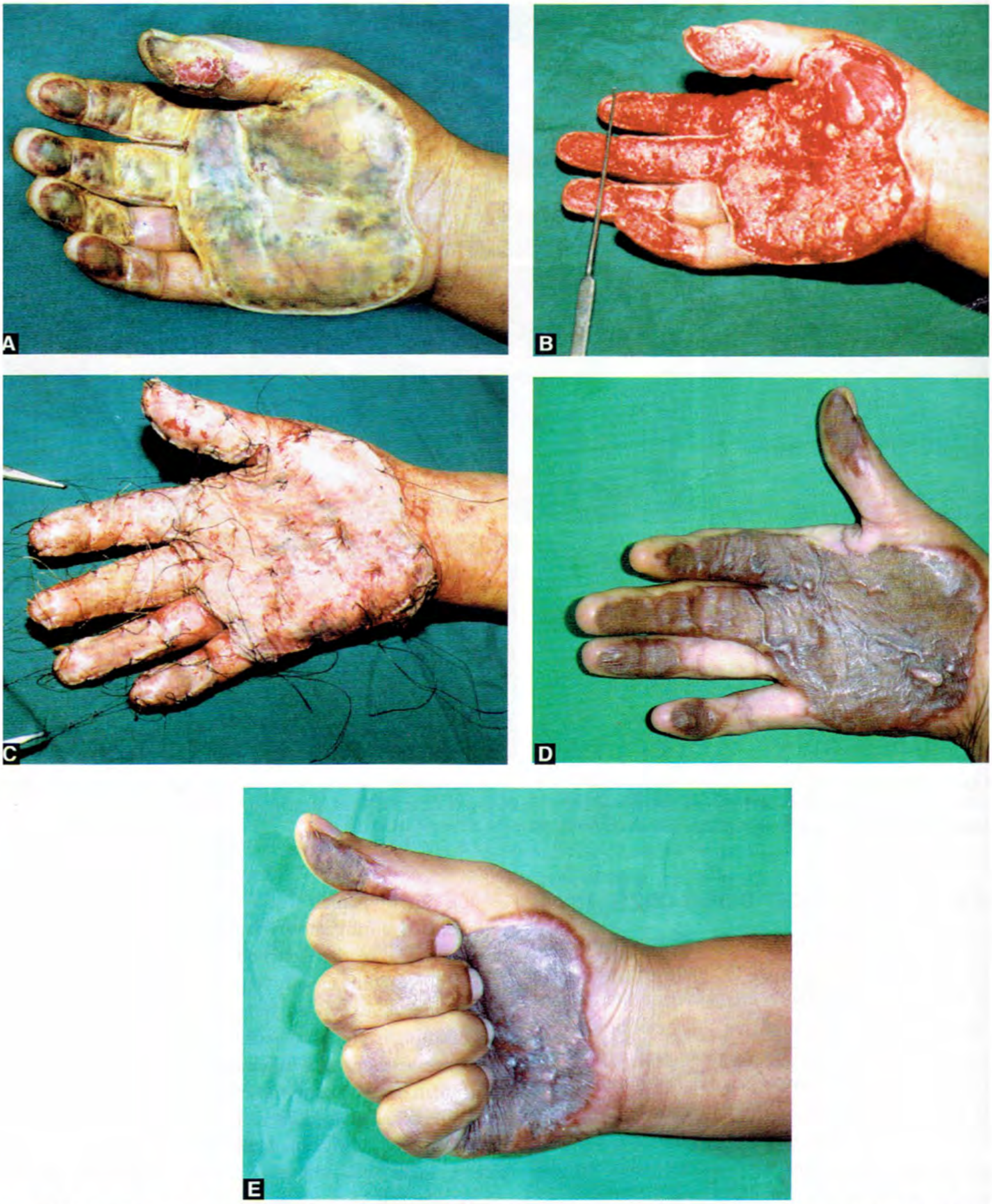
Figs 27.8A to J: A 3-year-old child was brought three weeks after electrical burns to the hand involving the thumb, index and middle fingers (A to C). The wounds were radically debrided and the bones stabilized by K-wire (D to F). Small abdominal flaps were given to the thumb and middle finger and the index raw area skin grafted (G). Good functional result achieved (H to J)

FLAP COVER TO THE DEEP BURNS OF THE HAND

In fourth degree burns, it is wiser to resort to early flap cover than just rely on positioning the joints. Various local, regional pedicle flaps like radial and posterior interosseous flaps, distant pedicle flaps and free flaps can be used to cover post burn raw areas (Figs 27.8A to J). Any of them can provide good result provided care is taken to keep the joints in functional position during flap transfer. Critical to the success of flap transfer is debridement of the area. The base has to be debrided to viable depth and the wound margins excised to good viable skin. This is crucial for success, for otherwise the flap detaches and slips off from the edges. A vascular flap cannot make the dead burned tissues in the base alive.

It is rare for palmar side to need flap cover. Palmar skin is thick and during most injuries the palm escapes deep burns because of the clenched fist position during injury. Deep palmar burns usually require flap reconstruction only when there is associated flexor tendon exposure or loss. Skin grafting is preferred because it produces the fixity and resistance necessary for satisfactory grip (Figs 27.9A to E). As with all hand burns it is essential to ensure total 'take' of the graft. When flap cover is required to the palm it has to be thin. Bulky flaps give a sense of fullness in the hand and prevent gripping of objects.

Reconstruction of severely burned fourth degree hand burns: Circumferential fourth degree burns may need amputation. In all other burn injuries all effort must be made to save available length of the hand. A very common pattern

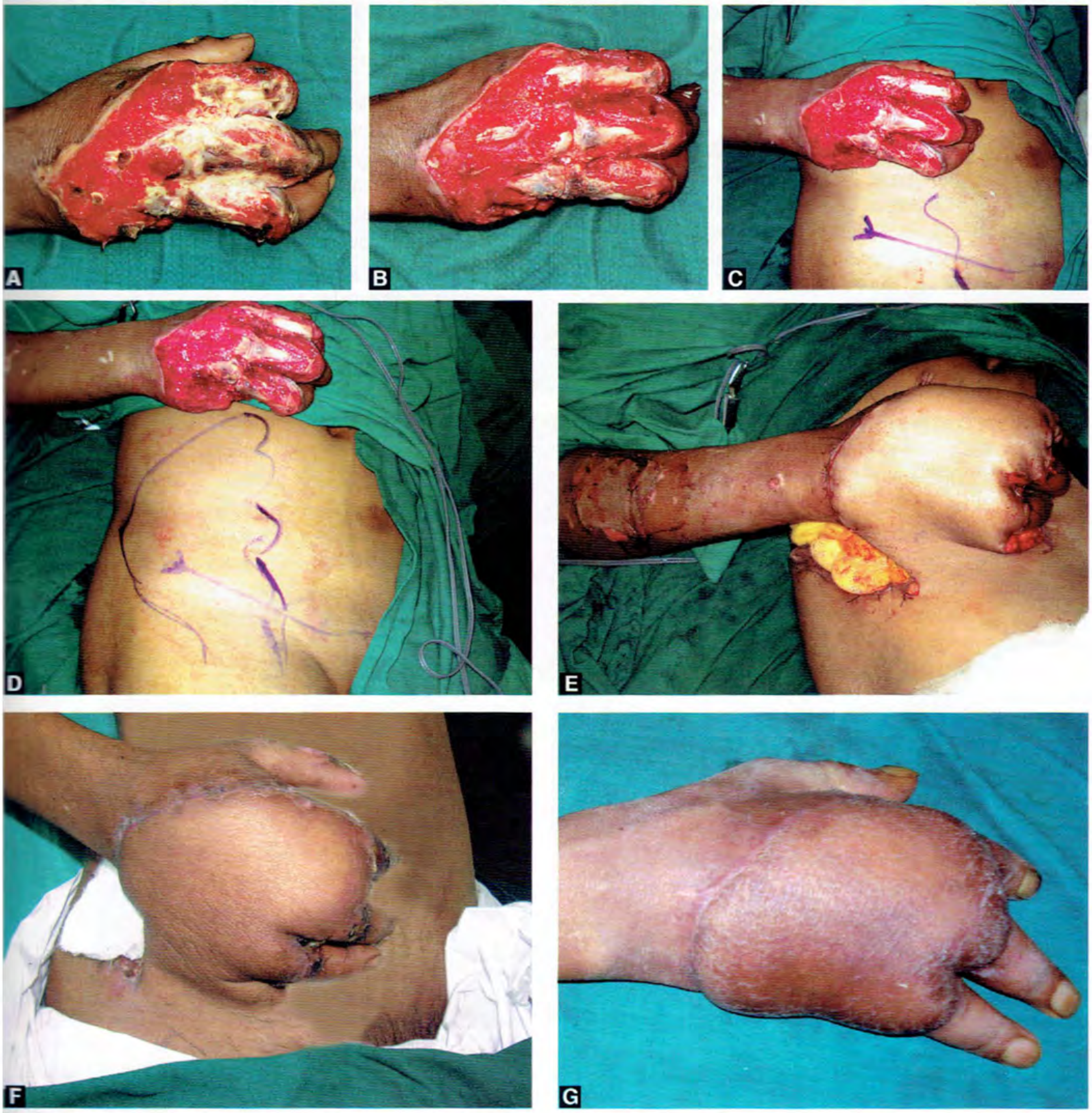


Figs 27.9A to E: Deep burn of the palm sustained during an epileptic attack (A). The area was excised and covered with split skin (B and C). Good after care with compression garment and massage gave satisfactory outcome (D and E)

of injury is very deep dorsal injury exposing burned bone with viable volar side (Figs 27.10A to 27.11K). Advances in flap cover and microsurgery can make these hands functional. Early flap cover rather than simple K wire stabilization is advocated for salvage of these hands.[11]

AFTERCARE OF BURNED HANDS

Totally healed burned hand is a good platform to start rehabilitation. Even small areas of unhealed wounds give rise to inflammation, need regular dressing, are painful and make the patient uncooperative for rehabilitation schedules.



Figs 27.10A to G: Deep burns involving the dorsum of the hand with loss of extensors, exposed joints and phalanges (A). The wound was well debrided (B), and an abdominal flap planned including both the groin and hypogastric territories (C to G). The flap was subsequently thinned. He had reasonable function of the hand even without extensor tendon reconstruction

The aim is to minimize the edema, increase the range of movement and prevent scar hypertrophy. Daily supervised physiotherapy is essential.

The value of pressure to prevent hypertrophy of scar was advocated by Parks [12] and Larson.[13] They advocated continuous controlled pressure of the magnitude of 25 mm Hg above the capillary pressure. Custom made compression garments are prescribed and the patient is made to wear it at all times other than at the time of exercises. They must be worn for at least 6 months. These are most effective in the early stages and hypertrophic scar is responsive to pressure in the first 3 months (Figs 27.12A to H). For this reason it is emphasized that the end point of acute burn management of the hand is to get stable healing of all the wounds. Patient compliance with pressure garments is not good if they are worn over dressed wounds. The bleeding from the wound and the pain deter them from wearing the pressure garment and ranging of the joints. Hence it is our practice to readmit the patients even when there is a small area of skin loss and re-graft to get good stable healing. It is common for contractures to occur in the web. One of the effective ways to prevent it is to roll the micropore plaster, place it in the web and hold it by tapes (Fig. 27.13).[14]

Perkins[15] initiated the use of silicone gel in scar for controlling hypertrophic burn scars. Though the mode of action of the gel is still uncertain, clinically it has been found to be effective. Probably they act by decreasing the evaporation of water from the skin and maintaining the optimum hydration of the stratum corneum. Like pressure garments it also has to be applied for a few months. Silicone gel application allows early pain free movements of stiff joints. When applied over healed skin grafts it prevents contraction of the grafts and when applied over hypertrophic scars, the scars soften and make them more amenable to pressure therapy. It is the practice of the author to use both silicone gel sheet and pressure garment together whenever possible in conjunction to get optimum results. Silicone product improvement has been done and now it is available in a tube as a gel. It has the advantage of easier application in areas of varying contour and patients find it easier to comply. Equally good results have been claimed.[16]

It is to be emphasized that prime consideration in the prevention of hypertrophic scar and contracture is to obtain early and complete healing of the burn wound in the hand.

POST BURN HAND DEFORMITIES AND THEIR MANAGEMENT

Complications of hand burns are due to poor wound healing, namely hypopigmented scars, hypertrophic scars and joint contractures. They cause both functional and esthetic problems.

Hypopigmented areas are due to delayed wound healing and are areas which would have benefitted from early grafting. If cosmetically disfiguring, the areas are excised and covered with partial thickness skin graft. Complete excision has to be done as otherwise remnants at the rim give poor outcome and are difficult to treat.

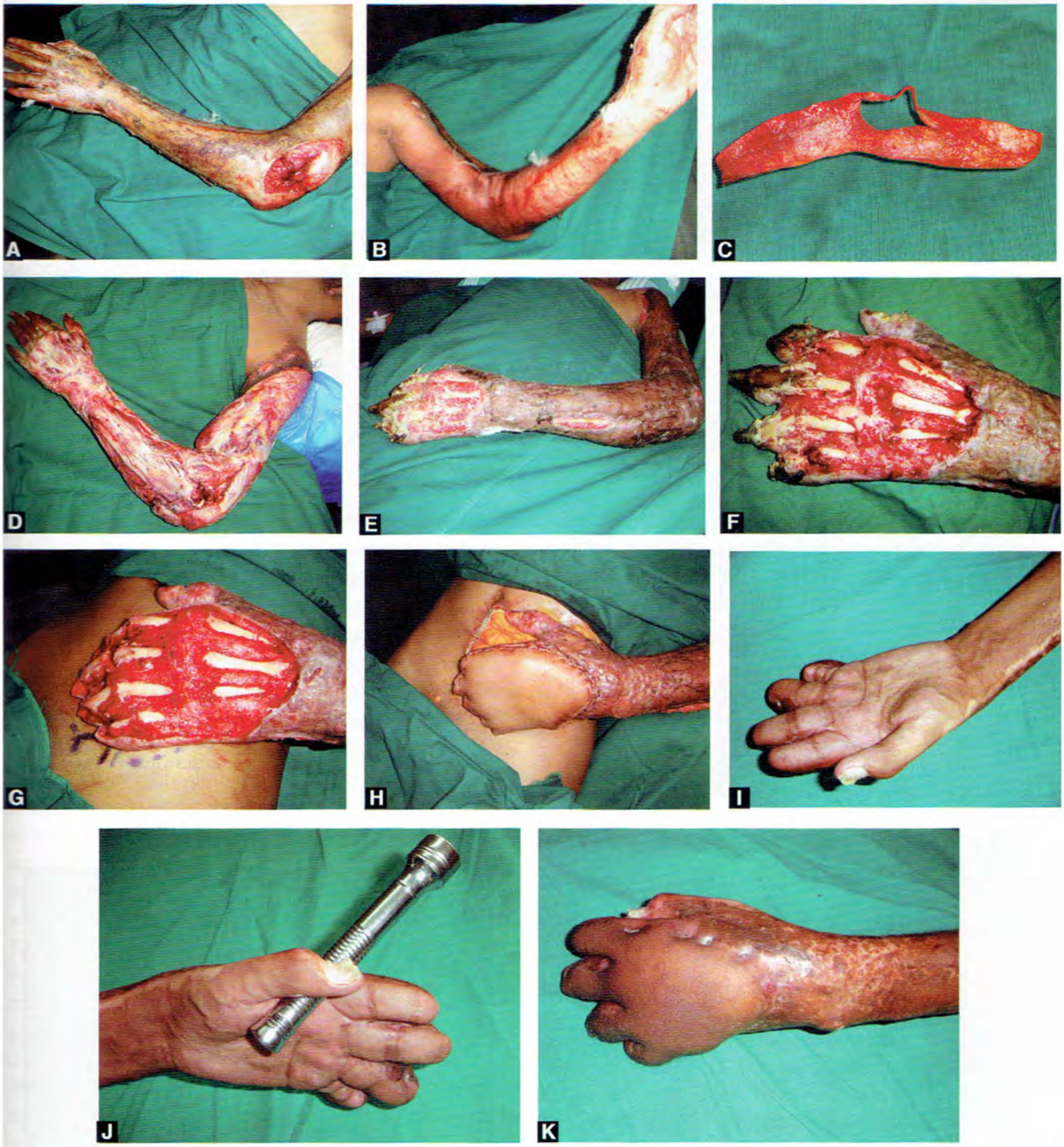
Hypertrophic scars again are due to delayed wound healing and occur in areas which would have benefitted from early skin grafting. They may be associated with contractures. Release of contractures by incising the hypertrophic scars and grafting will release the tension in the scar. Initially the variations in contour may appear unsightly, but aftercare with compression garments allows it to settle down and provide quite acceptable results.

Contractures result in great functional problems. McCauley[17] proposed a classification system which correlates the severity of burn scar contractures to hand function (Table 27.2).Contracture release is done when the scar equilibrium has been obtained which is evident by absence of scar tenderness and redness. This usually occurs in 3 to 6 months. Recurrences after release are less common when release is done at this period. It is usual to obtain the release of axilla and elbow contractures before the hand contractures are dealt with. Technical aspects of release of some common joint contractures are dealt with in detail.

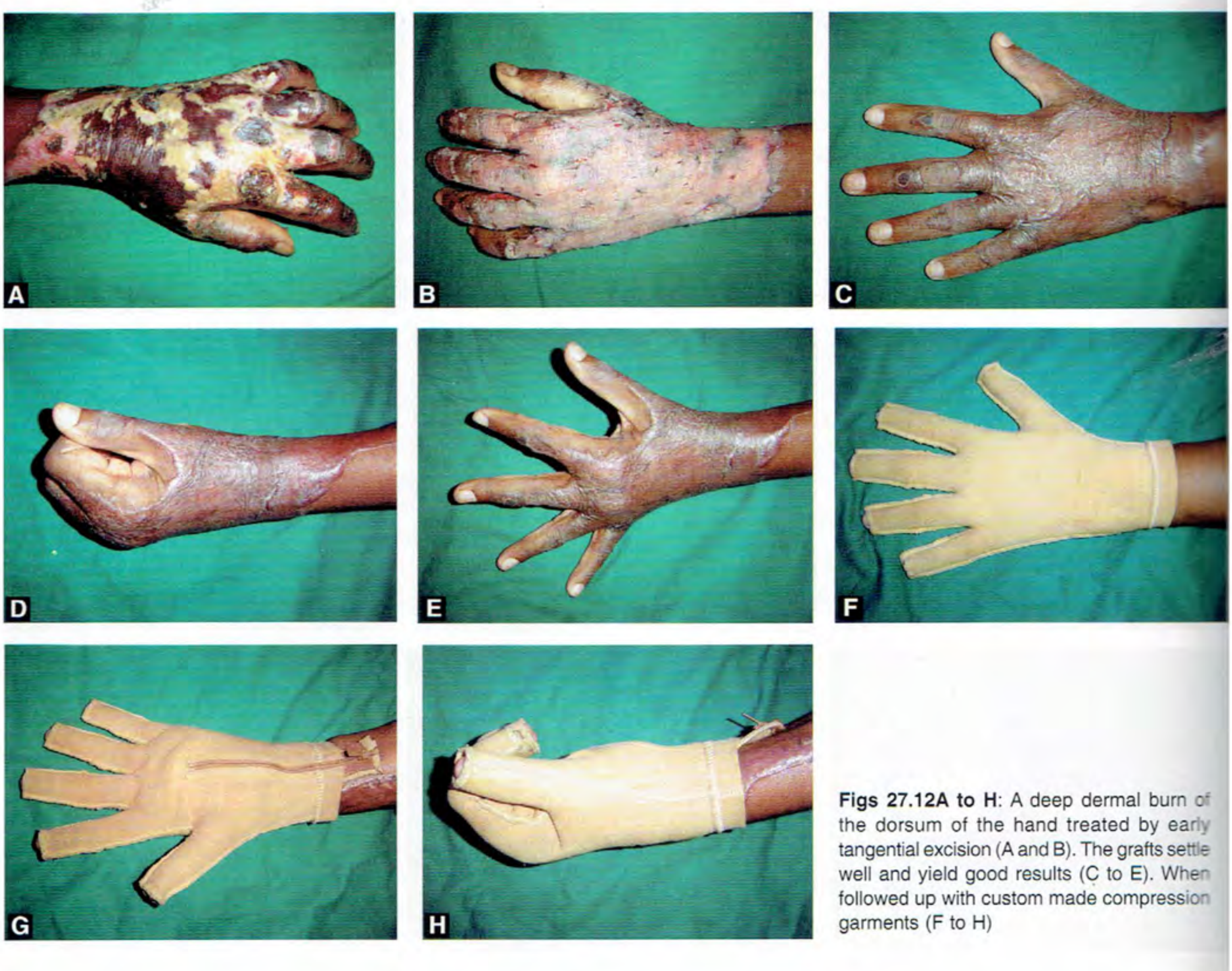
Metacarpophalangeal Joint Contractures: An MCP joint contracture can be in extension or in flexion. A well functioning MCP joint along with a good first web are the cornerstones for obtaining good function in the hand. When releasing extension contracture an assessment is made about the type of skin cover needed. If flap cover is to be provided

Table 27.2: McCauley's[17] classification of burn scar contractures in hand

Grade I	Symptomatic tightness but no limitation in range of motion, normal architecture
Grade II	Mild decrease in range of motion without significant impact on activities of daily living, no distortion of normal architecture
Grade III	Functional deficit noted, with early changes in normal architecture of hand
Grade IV	Loss of hand function with significant distortion of normal architecture of the hand
	Subset classification for Grade III and IV contractures A: Flexion contractures B: Extension contractures C: Combination of flexion and extension contractures



Figs 27.11A to K: Fourth degree burns of the dorsum of the hand, forearm and arm with relative sparing of the volar side in an industrial accident (A and B). The dead skin was excised (C), which revealed the damage to the muscles (D). The wound was serially debrided and the arm and forearm were grafted. The hand was subsequently debrided and covered with a groin flap to achieve a satisfactory functional outcome (E to K)



Figs 27.12A to H: A deep dermal burn of the dorsum of the hand treated by early tangential excision (A and B). The grafts settle well and yield good results (C to E). When followed up with custom made compression garments (F to H)

an incision can be made over the MCP joints to obtain release. Otherwise a transverse incision is made 2 cm proximal to the joint line and flap raised. After contracture release the distal skin migrates to cover the joint while skin graft is applied proximally (Figs 27.14A to D). Collateral ligament release and pericapsular dissection may be needed to obtain full release. Two major problems exist in MCP joint extension contractures which need attention. One, the contracted collateral ligament needs release (Capsulotomy). Second, a pocket needs to be created on the volar side for the base of the proximal phalanx to slide down the head of the metacarpal. For capsulotomy, the capsule is entered by making an incision over the extensors and by gentle sweep of a No.11 blade the contracted collateral ligament is incised. When released it is possible to flex the proximal phalanx but without the pocket in the volar side, the joint subluxates

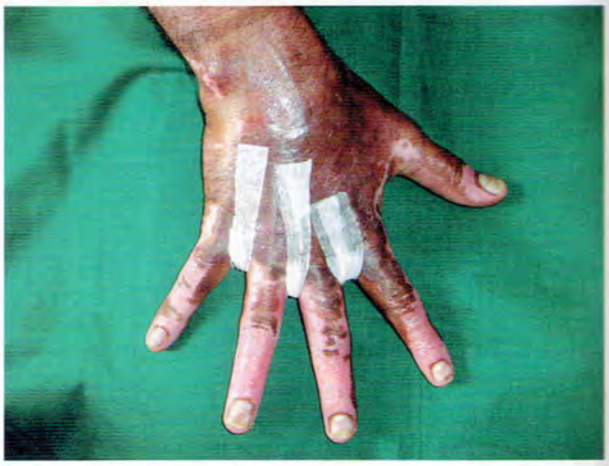


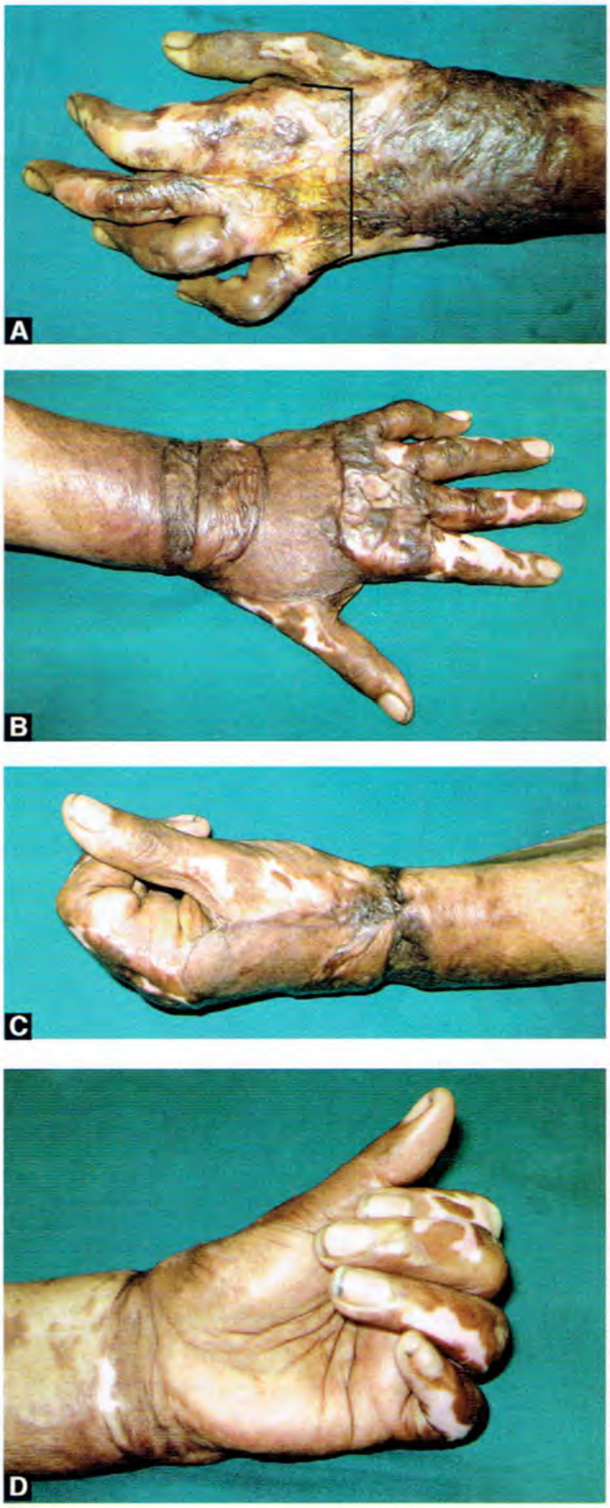
Fig. 27.13: To prevent web creep after grafting of the web spaces, compression is needed. It can be obtained by placing a rolled micropore plaster retained by a tape

(Figs 27.15A to C). This needs to be taken care of. By a combination of usage of curved scissors and curved dissectors, the adhesions on the volar aspect of the MCP joint are released. Proper translation of the base of the proximal phalanx over the head of the metacarpal is confirmed both clinically and radiologically. If there is resistance for maintaining the position, the joint should be pinned and most often, flap cover is required in such cases.

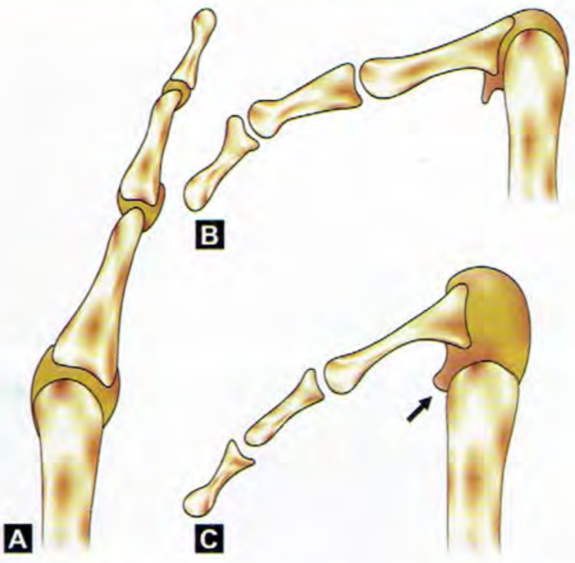
Extension contracture of the MCP joints is frequently associated with flexion contracture of the IP joints. Release of the MCP joint may make the finger tips bury into the palm. Usually it is difficult to correct all the contractures at the same time and this temporary problem has to be explained to the patient beforehand. If the PIP joint contracture needs volar release, it is done in another sitting. For some cases, arthrodesis of the PIP joint may be the chosen option and in that case both the MCP and the PIP joint releases can be done at the same time.

Proximal Interphalangeal Joint Problems: Although regaining of movement in all the joints is the aim of every hand surgeon, in burned hands particularly at the PIP joints, arthrodesis may be one of the common operations resorted to. This is because of the original damage to the extensor apparatus that occurred at the time of injury or subsequently due to edema, poor positioning of the joint and loss of skin over the PIP joint. It is best to prevent the problem since an established PIP joint contracture is resistant to treatment. In the acute situation the PIP joints are splinted in extension. If there appears to be a risk of skin loss over the joint an oblique K wire is passed through the joint to keep it in extension. This reduces the stress on the skin. It may heal or else granulate enough to be covered with skin graft. This step may prevent loss of extensor tendons and joint exposure. This is a crucial step in the care of hand burns. Otherwise, the central slip ruptures and the lateral bands slide down resulting in a boutonnière deformity. If the joint is exposed early debridement and cover with local flap is tried. If not, distant flaps have to be resorted to. In isolated hand burns this is practical, but if this problem occurs in a major burn the joint may be lost in debridement and the wound heals with the finger shortened.

Boutonnière deformity is due to the attenuation or rupture of the central slip and the sliding volarwards of the lateral bands. The deformity has two components – flexion at the PIP joint and extension at the DIP joint (Figs 27.16A and B). If a boutonnière deformity is passively correctable, the



Figs 27.14A to D: For the release of dorsal contractures, an incision as in the figure will slide the distal flap to cover the joint after release. Joint release can also be done under the flap. If the release process does not expose critical structures skin graft may be applied to the raw areas



Figs 27.15A to C: During the release of the hyper extension contracture of the MCP joints of the fingers, it is important to clear the tissues on the volar aspect of the head of the metacarpal and create a pocket (A and B). Otherwise the joint subluxates (C) and if pinned in that position the contracture will recur

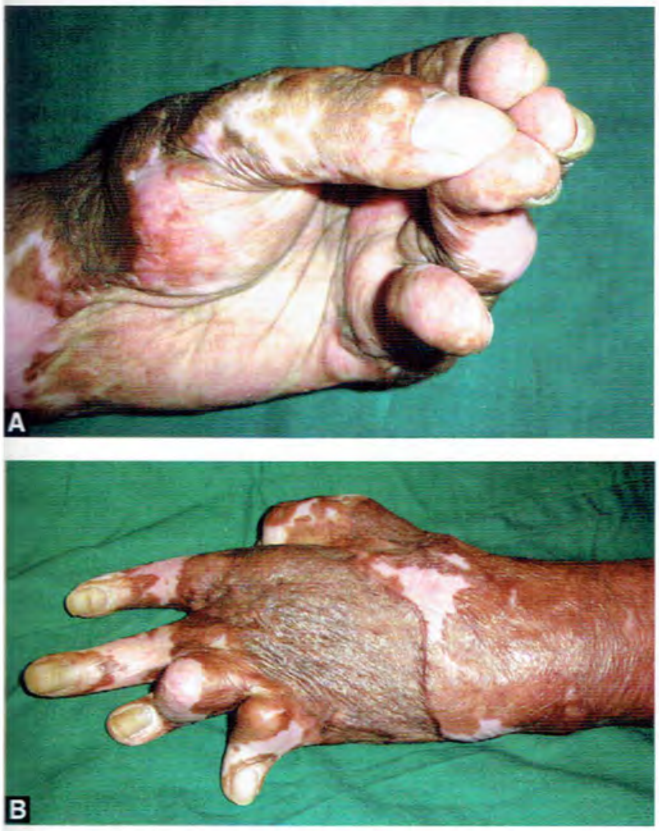
PIP joint is splinted in extension and the patient is encouraged to flex the DIP joint. The flexion of the DIP joint in turn makes the lateral bands slide up at the PIP joint level. Therapy is very important in boutonnière deformity correction and no surgery is likely to succeed unless both the components of the deformity are passively correctable. If passively correctable and the dorsal skin overlying the PIP joint is of good quality,correction can be attempted by reinserting the central slip, freeing the lateral bands from the volar side and plicating them dorsally. The limiting factor is the skin condition over the joint which usually is of poor quality. So arthrodesis of the PIP joint is the commonly advocated procedure.

The aim of arthrodesis of the PIP joint is to bring the joint in a position so that it can achieve good contact with the thumb pulp. If stable fingers achieve pulp to pulp contact with the thumb, the hand becomes functional. To this end, the recommended angle of arthrodesis of the PIP joints are 25-35° for index, 30-40° for middle, 35-45° for ring and 40-50° for little finger. This angle may be varied according to the length of the finger and the movement possible in each of the components. Technically, a few points are worth noting. It is better to access a too much flexed joint by a gentle curvilinear incision than a longitudinal incision. When a hyperflexed joint is arthrodesed, secondary to bone shortening which is part of the procedure, the incision may open up into a diamond making closure difficult. The flap is carefully raised and the joint entered by making a longitudinal incision in the midline. The collateral ligaments are divided by a gentle sweep of No.11 blade along the sides and the two articular surfaces are brought out to view. To obtain the correct angle of arthrodesis, one of the components is transected transversely and the other at the recommended angle of arthrodesis. The author finds this easier than trying to adjust the angle on both sides. K wires are used for stabilization. The bone is osteoporotic and fixation has to be done by single pass of K wires. As the fingers are immobilized, even a single oblique K wire is sufficient. The K wires are retained for a minimum period of 4 weeks and if the skin condition is good the wire may be retained for 6 weeks. The MCP joints are mobilized during this period.

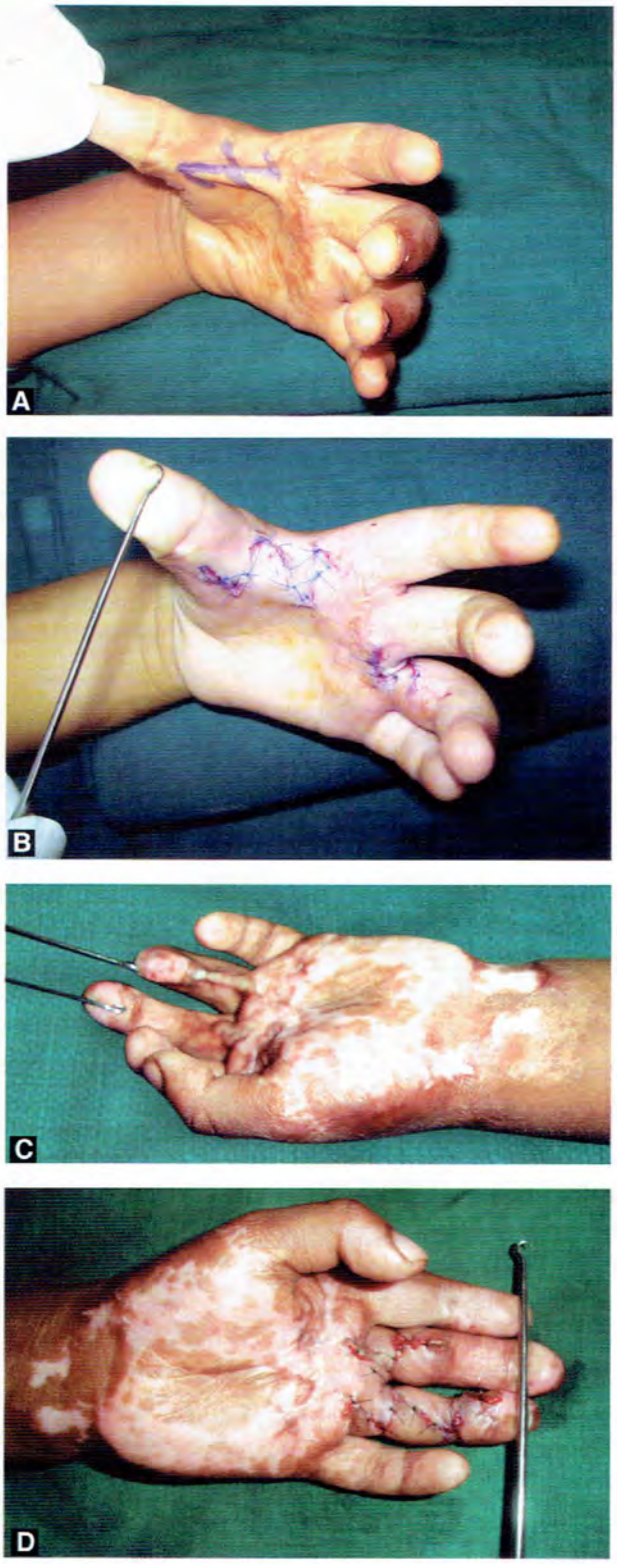
Distal Interphalangeal Joint Problems: Deep dorsal burns over the DIP joint cause attrition rupture of the extensor insertion resulting in a mallet finger. This can also, secondarily, lead to swan neck deformity. Mallet finger and swan neck deformities are functionally more disabling than boutonnière deformity. Due to poor quality of the skin overlying the joint, tendon reconstructions are usually not possible. Arthrodesis of the joint in 0-5 degrees is done by wedge resection of the articular surfaces. Performed early, it may prevent the development of swan neck deformity. In burns, the swan neck deformity is more often due to scar hypertrophy and contracture of the dorsal skin of the fingers. In these situations, incision release of the hypertrophic scar in two places preferably over the proximal and middle phalanx and grafting the residual raw areas is very useful. When the hyperextension at the PIP joint is addressed and the DIP joint remains flexed, the deformity becomes non-functional. The DIP joint then needs to be arthrodesed in 0-5 degrees.

Web space problems: *First web contracture*—The problems of contracture of the first web are different from the contracture of other web spaces. While web contractures in the fingers affect the individual mobility of the fingers and the ability to spread, first web contracture affects the span of the hand. It reduces the working space of the hand and is functionally more crippling.

In the acute phase, the general tendency of the thumb is to lie in an adducted and extended position. Left as such, the narrowing of the first web occurs and thenar muscles and joints get stuck in that position. The contracture may be due to the tight skin or may be associated with intrinsic contracture and joint contracture. If it is due to the skin only, the contracture can be corrected by Z plasty or its various modifications (Figs 27.17A to D). Frequently, it needs further release and in such situations, the author prefers to extend the dorsal limb of the skin incision proximally to access the deeper structures. The fascia over the interosseous and the adductor is incised.The Z plasty flaps are transposed. This provides good skin in the web and the defect on the dorsal aspect of the web is skin grafted (Fig. 27.18). If the contracture is more severe, the first dorsal interosseous muscle is released from its origin at the first metacarpal and the adductor pollicis origin is shaved off from the third metacarpal. This enables them to retain their blood and nerve supply. The badly contracted first



Figs 27.16A and B: Boutonnière deformity in the ring finger (A). Correction is difficult because of poor quality skin on the dorsum. In spite of the deformity function is preserved in most cases



Figs 27.17A to D: Z plasty and its modifications are good for the release of band contractures (A and B). When done in the fingers bigger Zs are better than small flaps (C and D)

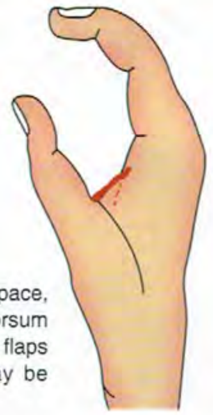


Fig. 27.18: For release of the first web space, a Z plasty incision extended on the dorsum gives good exposure. The transposed Z flaps cover the web and the raw area may be covered with graft

web needs further release at the first CMC joint. Only then the web opens up. While doing this, one has to be careful not to damage the radial artery which passes from dorsal to the volar side close to the joint. The released web is maintained by a K wire spacer and a flap cover is provided. As the local flaps are generally not available, a posterior interosseous flap or a distant groin flap has to be used (Figs 27.19A to J).

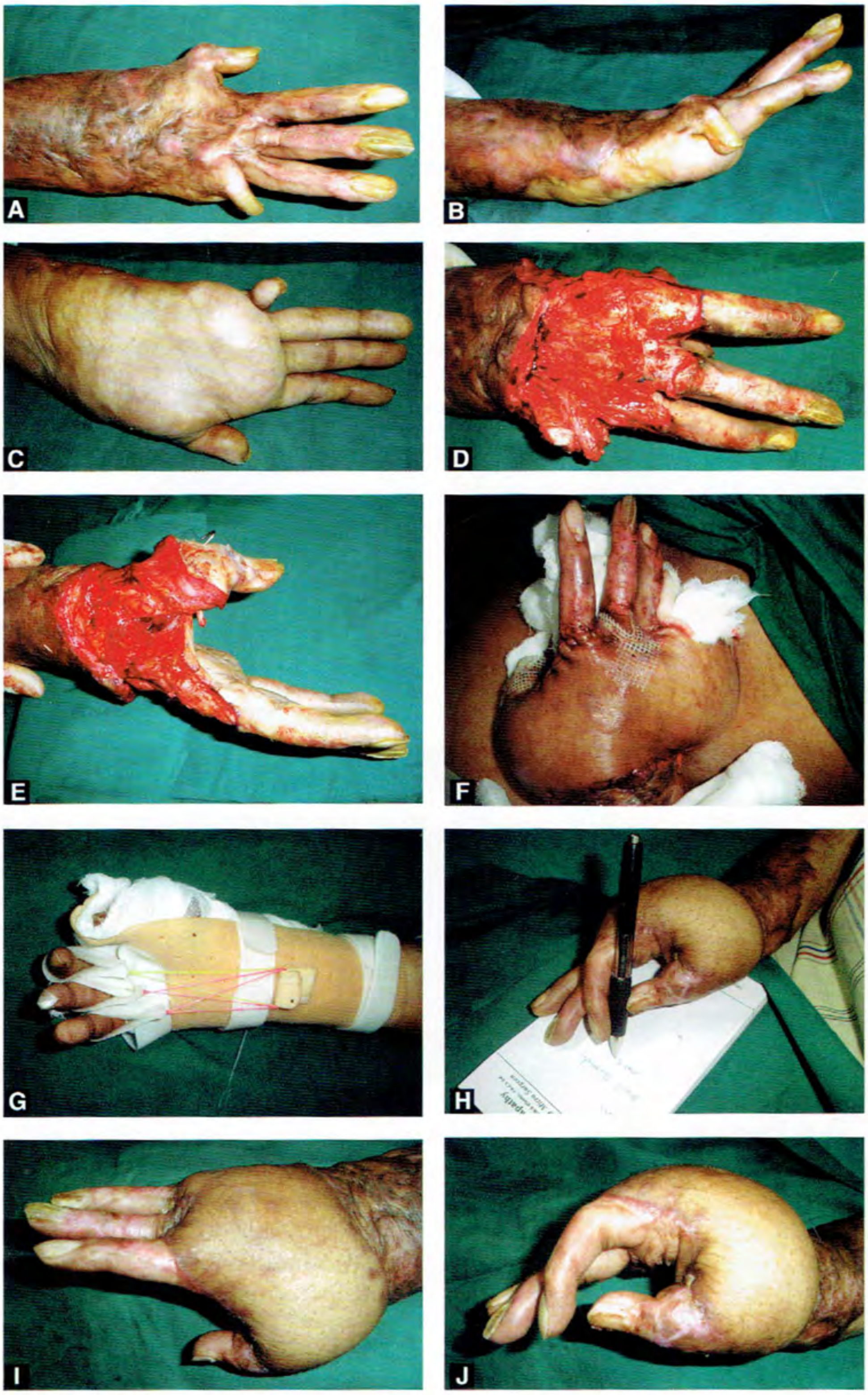
Post burn syndactylization of fingers: Fingers may need syndactyly correction or correction of the web creep. Minor web contractures can be corrected by Z plasties –four flap technique or V-M plasty. More recently the author is using the square flap technique which is also useful for more severe syndactylization (Fig. 27.20). It is important to achieve good primary healing of the wounds after surgery so that pressure garments and silicone gel compression can be instituted early. Physical therapy is the corner stone for the prevention of web creep.

Volar Contractures: Contracture of the volar aspect of the fingers is more common than that of the palm. Little finger is more often affected and more severely than other fingers and correction is also more difficult. Incision release is done for these contractures and the resulting raw areas are preferentially covered with full thickness skin grafts. Sometimes the volar plate and the joint capsule need release. In such situations the joint needs stabilization with a K wire till the skin cover settles. Limiting factor for release is distal vascularity. After obtaining the release possible, the tourniquet is released and vascularity of the fingertip is checked. If it is slow to pick up, the joint is gradually flexed to the extent where the fingertip turns pink and the joint fixed at that level. Checking the vascularity of the fingertip is important at the completion of contracture release and in the immediate postoperative period. If joint release is done or the flexors and the nerves are exposed it is preferable to provide flap cover. Cross finger flap is a common flap used in this situation (Figs 27.21A to C). The author has a low threshold for using flaps on the volar aspect of the fingers after contracture release. In marginal situations if skin graft is applied over the flexor sheath and digital neurovascular bundles, the graft may be expected to 'take'. In the event of recurrent contracture during revision surgery chances of injury to the digital neurovascular bundles is very high.

Color of the skin graft on the volar aspect of the fingers is a cause for concern for some patients. If the raw area to be covered is small, second layer of palmar graft from the hypothenar eminence is a good source for a better color match; other sources are grafts from the instep area. The morbidity at the donor sites and the limited area of skin available are factors against their common use. Skin grafts, in course of time, become supple and the patient acceptability increases with time.

Amputations: Finger and hand amputations occur in circumferential fourth degree burns and in major electrical burns injury. While it is unavoidable in such situations, amputations must not be allowed to occur due to infective complications. Functionality of the hand depends upon the capability of the thumb stump to meet the tips of fingers. Length of the remaining fingers, the depth and the span of the first web are the limiting criteria of function. Following salvage procedures are possible.

Phalangisation is one of the early procedures recommended for the reconstruction of the mitten hand. This involves deepening of the first web, receding the adductor insertion proximally and excising the index ray remnant. There is an increase in the first web space at the expense of the palm width.This was succeeded by *Pollicization* wherein the index ray remnant is transferred over the thumb stump.[18] This is a technically demanding procedure particularly if the web skin is intact. If the index stump has some sensate volar skin it is transferred with the nerve. In both techniques the result depends upon the intact thenar musculature and the length of the remaining stumps. The third alternative is *distraction lengthening* of the stumps. If the whole of the first metacarpal is intact, distraction



Figs 27.19A to J: A severe contracture of the hand (A to C). Release requires complete excision of all scar tissue (D). Creating a good first web is important for function and is retained by a K wire (E). A groin flap cover is given (F), and aggressive physiotherapy is essential (G). Satisfactory functional outcome can be achieved (H to J)

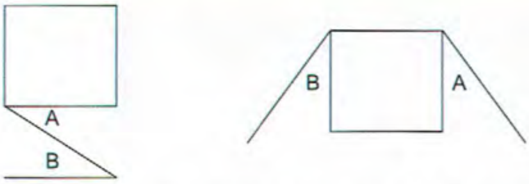
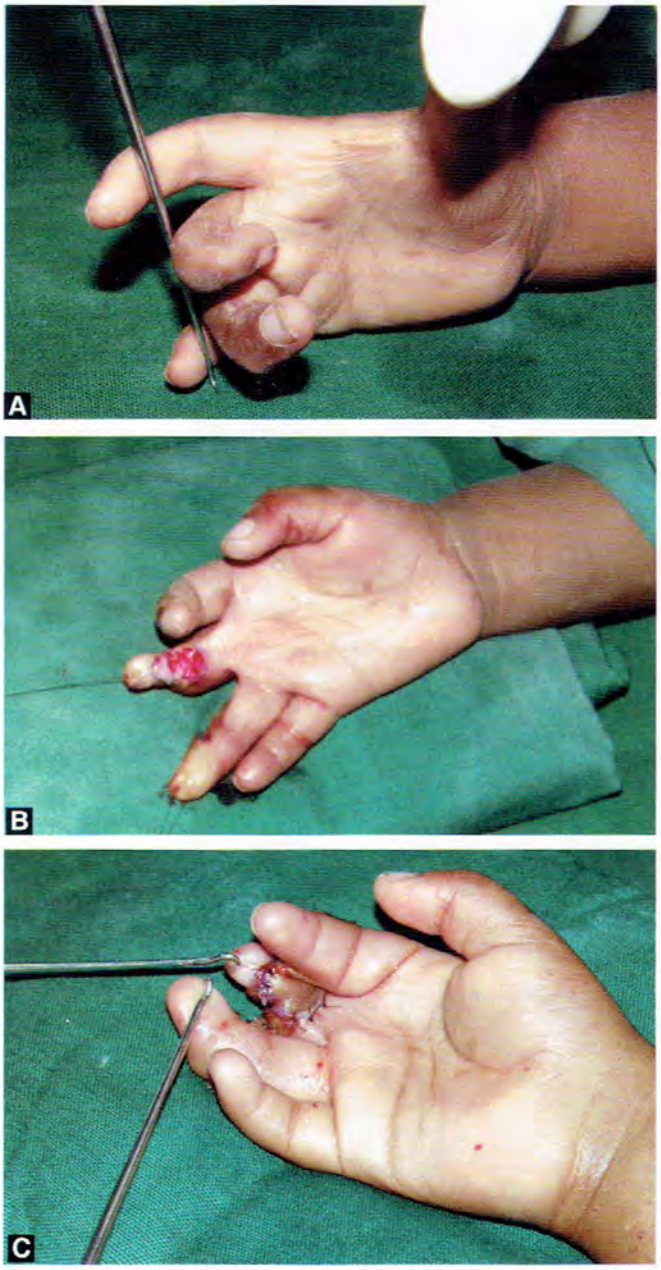


Fig. 27.20: Square flap technique for deepening of the web



Figs 27.21A to C: Volar contracture in the middle finger covered after release by a cross finger flap

lengthening as first advocated by Matev may be used. Subperiosteal transverse osteotomy is done in the middle third of the first metacarpal and distraction of 25 mm can be achieved. Skin quality must be good both at the stump and in the line of the pins. Otherwise prior flap cover is advised.

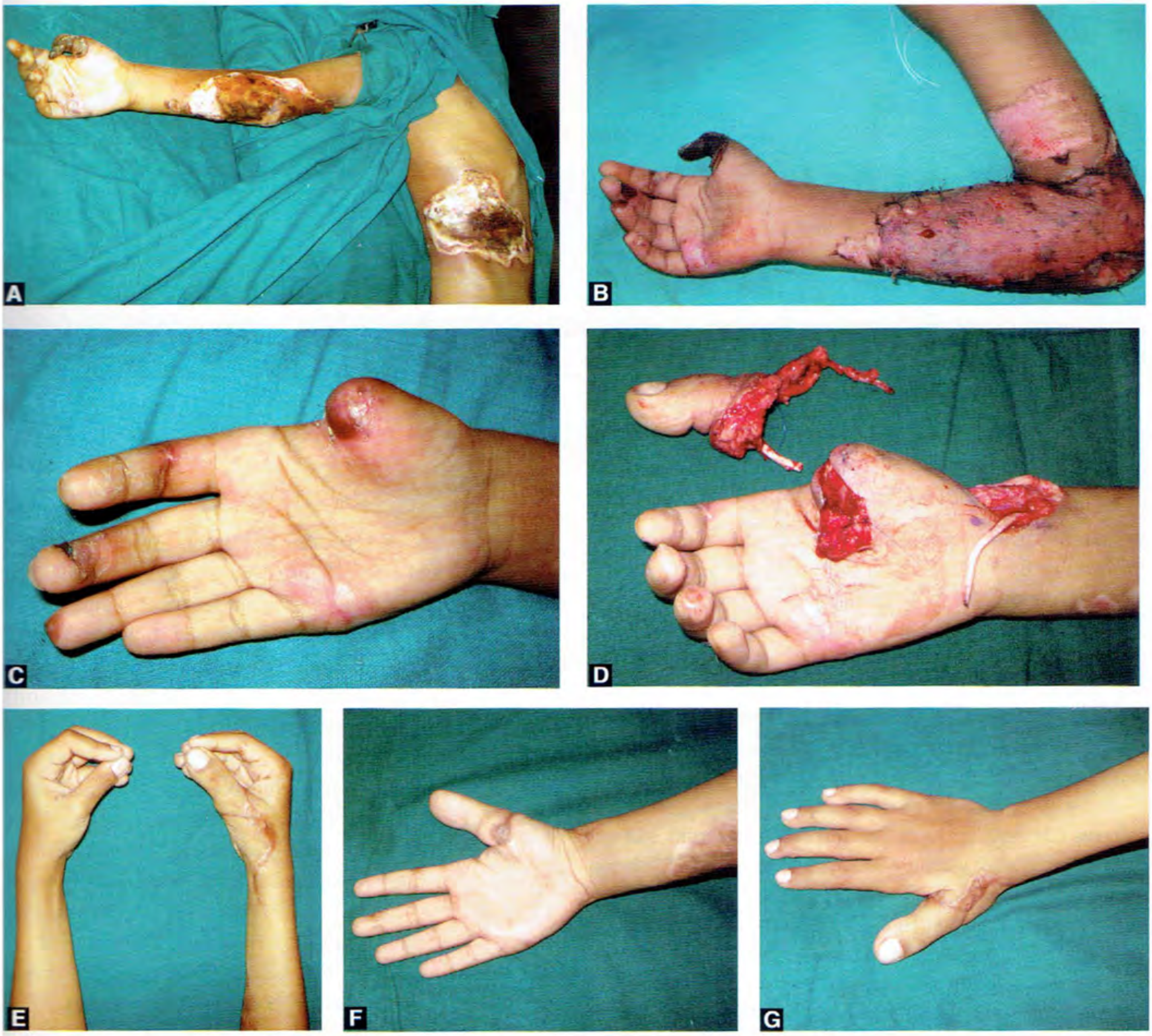
Advent of microsurgery has increased the options of reconstruction. The most valuable is *toe transfer*. This procedure gives a good functional and cosmetic result in one stage. This is the best possible reconstruction in isolated thumb and finger losses due to electrical burns and work place burn injuries. In children, it has the added advantage of retaining the growth potential (Figs 27.22A to G).

The last alternative is *osteoplastic thumb reconstruction*. Though the literature is abounding with articles against it, the author finds it as a very valuable technique for a person who is unwilling to sacrifice the toe. When done well, the results of osteoplastic thumb reconstruction matches in function with other types of reconstruction.[19] Osteoplastic thumb reconstruction must be used only if there is a potential for performing the neurovascular island flap to provide sensation. Otherwise bone resorption rate is high. This is a limiting factor in the use of this technique in post burn reconstruction.

REHABILITATION ISSUES IN HAND BURNS

Helms[20] studied the time to return to work following hand burns in 70 patients in relation to several variables. 1. Total body surface area (TBSA) burned, 2. Hand burned, 3. Grafting, 4. Patient's age, 5. Occupational category. When assessed at 8 months, 74% had returned to work. The best predictor of time to return to work was TBSA burned, followed by 'grafting' and the 'hand burned'. No significant differences or predictors were found for patient's age, occupational category or between non burn hand trauma patients in prior reports.

The sight and condition of the burnt hand is frightening to the conscious burn patients. It produces doubts and fears about their future in the work place and possible dependence on others for day-to-day existence. Along with good treatment, presence of reassuring physicians and therapists goes a long way to allay the fears and boost confidence in the patient. It encourages them to comply with the exacting schedules of physiotherapy and look up to a good future.



Figs 27.22A to G: A 5-year-old child with electrical burns involving right upper limb and abdominal wall (A). The thumb necrosed and the rest of the raw areas were skin grafted (B and C). Two months after the injury, thumb reconstruction was done with great toe transfer (D), with excellent functional result (E to G)

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Chapter

28

Burns of Perineum and External Genitalia

Deepak Nanda

INTRODUCTION

The perineum is naturally protected by a deep furrow between the thighs, often getting spared from burns and other injuries. Burns of the perineum and external genitalia usually occur in patients with extensive burns although isolated injuries are not uncommon. According to Lund and Browder's chart for body surface area estimation, only 1% of the total body surface has been allocated to external genitalia and perineum. However, American Burns Association has classified burns of this region as major burns similar to those of face and hand. Burns of these areas can result in great psychological trauma which may be disproportionate to the percentage of total body surface area involved.

Michielsen et al (1998)[1] found an incidence of 2.7% for burns involving genitalia and perineum out of more than 4,200 patients admitted over a period of 15 years. One-fourth of them were children with a preponderance of males (2.4:1). Angel et al (2002) [2], reporting from Shriners hospital (Texas) catering to children only, found 76 patients out of a total of more than 2,300 over a period of 10 years, having burns of this region, again with boys outnumbering girls by 1.5 to 1. A study by Chicago Burn Centre[3] indicates that out of 1600 patients admitted for burns, 93 had involvement of genitalia and perineum (5.8%). Another study from Japan[4] revealed that the percentage of patients with involvement of genitalia and perineum was higher; about 21.5%. Severity of burn injury to external genitalia was more in males for obvious anatomical reasons.

ETIOLOGY AND PATHOGENESIS

The commonest cause of perineal burns is hot liquids or scalds which varied from 55% to 85%.[1,2,5] Second most common cause was the flame burns accounting for 24%, 30% and 7% respectively in these studies. Other common causes are electrical contact and flash burns and chemical burns. Some rare causes of burns of perineum and genitalia have been enumerated like alkali leak into baby diaper, ignition of collected spirit based cleaning agent on plastic sheet by diathermy, involvement of the area by blasts injuries and during brachytherapy for prostatic malignancy.

The burns of perineum and external genitalia involve people of all ages from infancy to elderly (86 years). Studies from USA have suggested child abuse where the 'ring sign' is supposed to be pathognomic and these children are usually branded for life. In the study from Japan, 'Sitz bath' has been blamed for burns of perineum in old people especially during the winter months when these baths are a custom (Figs 28.1A and B).

Abdel-Razek[6] reported cases of rare isolated chemical burns of genitalia and perineum from King Saud Hospital. These burns constituted about 3.4 percent of total burns at his center. They were due to sulfuric acid used for water closet cleaning which was accidentally used to wash genitalia as an initial step of making Wodoa before praying in Islam. Sulfuric acid and its precursor sulfuric trioxide are strong acids and desiccants which cause coagulative necrosis of parts involved. These burns are either second degree deep dermal or third degree full thickness, the latter being more common (Fig. 28.2).

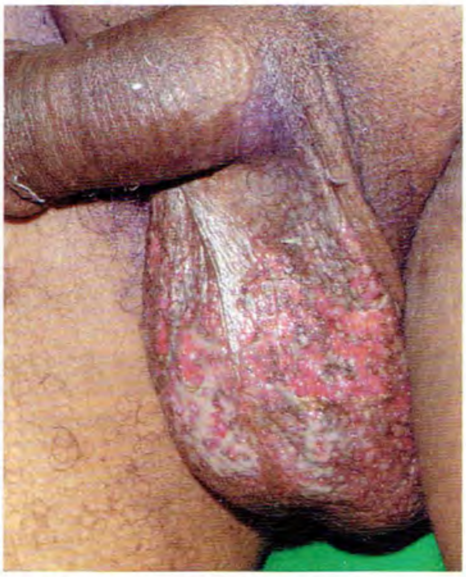

Fig. 28.1A: Accidental superficial burns of scrotum and perineum by concentrated potassium permanganate during sitz bath

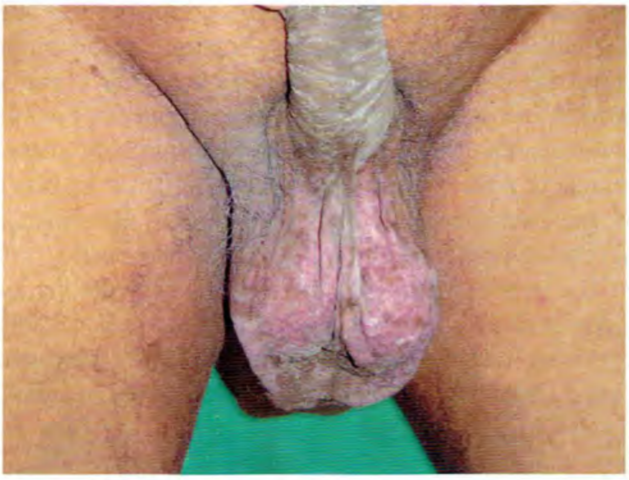

Fig. 28.1B: Same patient after 1 week with healed areas showing hypopigmentation

Rare cases of genitalia and perineal burns have been reported due to fireworks during Guy Fawkes nights and a case has been reported where a mentally depressed woman inserted a rocket two inches into her vagina and ignited it while trying to commit suicide![7]

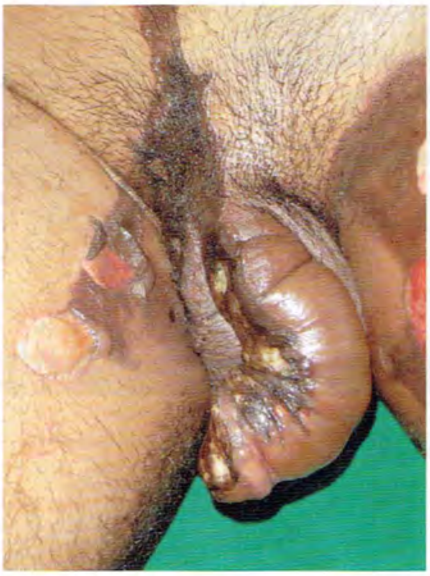

Fig. 28.2: Accidental acids burns showing track of acid trickling down from the abdomen towards perineum with deep burns over one side of penis causing lateral curvature with edema of penile skin

Burns due to scalds are usually second degree burns (Fig. 28.3). Out of these, superficial dermal are 1.5 to 2 times more common than deep dermal injuries. Third degree or full thickness burns constitute a small proportion only. Flame burns are usually deeper as compared to scalds (Fig. 28.4) while high voltage electrical contact burns may produce full thickness burns of perineum and partial or total loss of external genitalia (Fig. 28.5). Scalds are more common in children below two years while flame burns are commoner in the older age groups. Psychological consequences of these injuries cannot be ignored. Repeated examination of private anatomical area tends to affect modesty as well as self respect. Fear of sexual dysfunction and unattractiveness leads to depression. About 25 % adult patients complain of loss of libido and orgasmic dysfunction.[3]

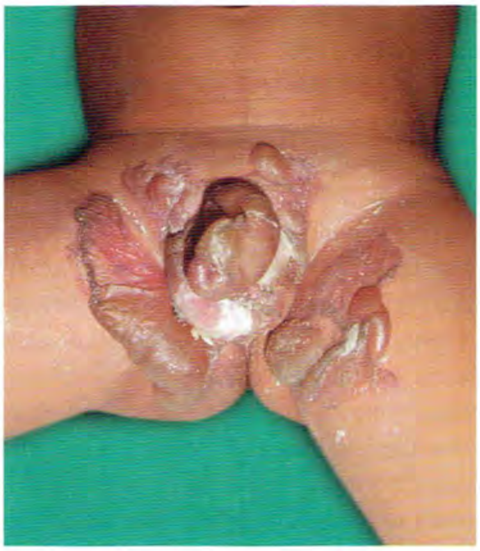

Fig. 28.3: A 10-year-old child with scalds of the perineum leading to superficial dermal burns with blister formation

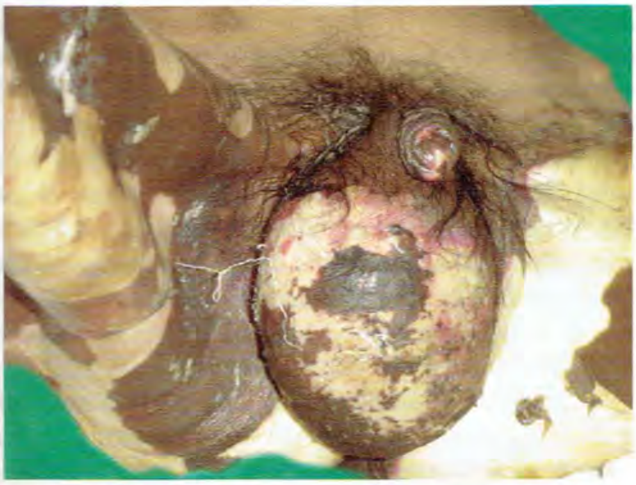

Fig. 28.4: Deep dermal thermal burns of scrotum and thighs

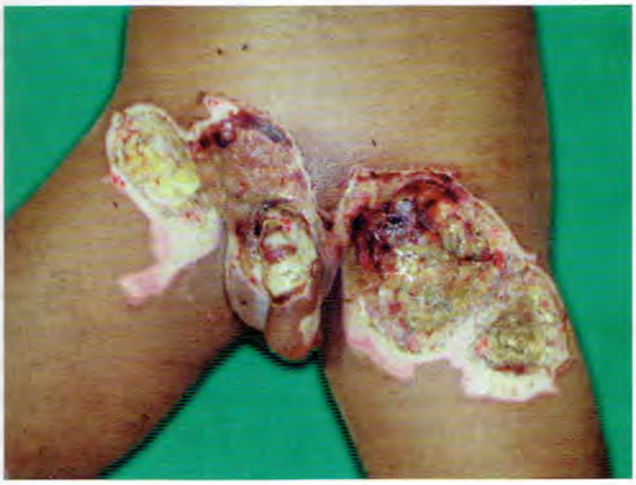

Fig. 28.5: Electrical contact injury causing full thickness burns in lower half of penis and scrotum and in groin causing exposure of femoral vessels on left side. Foley's catheterization possible in such cases

MANAGEMENT DURING ACUTE STAGE

Acute cases are managed by two different schools of thought viz: conservative and aggressive, the former advice only dressing of the involved area with antimicrobials so that bacterial count is kept below 10^5 and wait for the wounds to heal while the latter advocate early excision and resurfacing. What generally is followed in most centers is that superficial burns are managed conservatively or non-operatively with topical antimicrobials, small deep wounds of perineum are also treated conservatively as on healing they tend to contract and close rapidly by secondary intention. Eschar debridement, bacterial balance of less than 10^5 micro organisms per gm. of tissue should be maintained.

Urinary Diversion

Urinary diversion is usually required, catheterization suffices in most cases but suprapubic cystostomy may be required rarely (Figs 28.6A and B). They are usually restricted to the period of resuscitation to aid comfort and hygiene. Fecal diversions have been abandoned in most places but a study from Japan suggested temporary diverting colostomy for very old patients with perineal burns.[4] Urinary diversion is usually done by insertion of Foley catheter but rarely a suprapubic cystostomy may be required. Catheterization is usually required for about two weeks as most of the wounds heal by this time. Foley has additional advantages of preventing meatal stenosis and ammoniacal dermatitis. But catheterization is not without complications viz., urinary tract infection, urethral strictures following trauma during insertion and bladder stones. Rare complications like prostatic abscess, renal abscess, periurethral abscess, epididymo-orchitis and even urethral sloughing have been reported especially when catheterization has been prolonged. Thus, catheterization is required but should be minimized and monitored.

Prevention of Infection

Autoinfection of these areas is usually common due to proximity of the anus, particularly with *Pseudomonas aeruginosa*. Infection is dangerous as it destroys epithelial cells to convert partial thickness burns into full thickness burns, and also creates problems for graft 'take'.

Wound Management

Second degree superficial wounds of genitalia and perineum of both sexes and all ages are treated by exposure and application of silver sulfadiazine or other antimicrobial drugs. Other substances like potassium permanganate, sodium bicarbonate, etc. have been used but their efficacy is not proven. Daily baths and showers help in keeping the bacterial flora under control and separating the slough. Most partial thickness burns heal satisfactorily and even small full thickness burns can be managed by this method. Conservative management includes debridement of loose slough and application of topical antimicrobials. Broad spectrum parenteral antibiotics also form an integral part of the treatment plan. Open method of dressing is usually advocated as perineum and genitalia are difficult to dress. Most of the wounds heal satisfactorily by this method although a few may require secondary surgery for contracture release. Early mobilization is advocated for

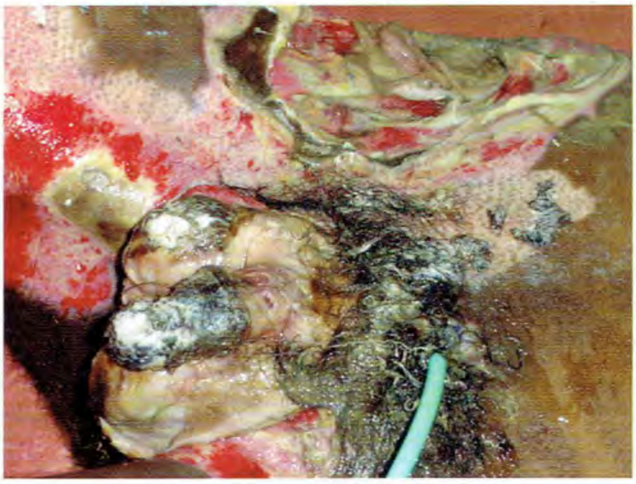

Fig. 28.6A: Electrical contact burns in perineum showing necrosed penis and scrotum with suprapubic cystostomy

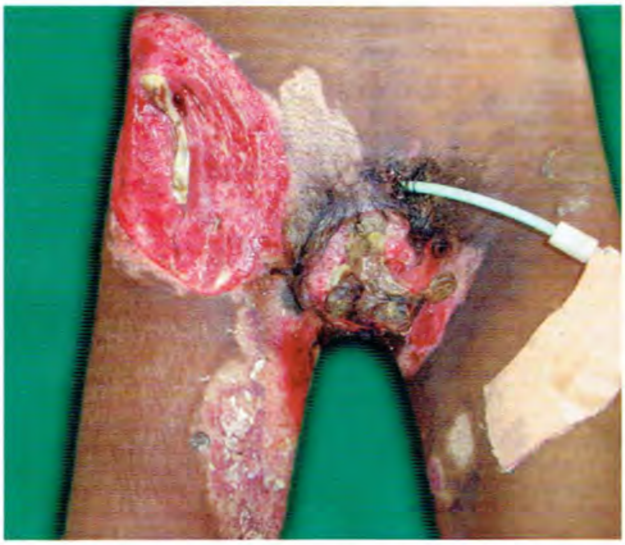

Fig. 28.6B: Same patient with total loss of penis and both testes

prevention of contractures. The deeper the wounds more frequent are the surgeries and more in number too.

For larger deep wounds surgical removal of devitalized tissue by tangential excision and skin grafting of the resultant wound is the main stay of management. Some authors believe that one should wait until eschar separates to maximize tissue preservation before one goes for grafting. Tissue preservation in this area is important as specialized tissue of this area is difficult to reconstruct. In the perineum graft should preferably be meshed in order to accommodate the irregular contour but expansion is not really required. Perioperative management of the graft is very important. The grafts require well vascularized bed with bacteriological balance. The graft requires stabilization with sutures, staples or fibrin glue. Post-operative shearing has to be prevented where a nylon mesh over the graft is helpful. Use of air ripple beds, nursing in prone position, minimization of fecal soiling decreases wound infection and graft loss. It has been observed that flame burns require more surgical intervention than scalds as the former are deeper. In one study, only 20% of scalds were classified as second degree deep or third degree while in contrast 66% of flame burns were of the same category.

When testes are involved, the burns are obviously full thickness as these are protected by skin, dartos, tunica vaginalis as well as cremasteric reflex. In such cases the thin penile skin usually does not survive. Early surgical intervention by excision followed by resurfacing with skin grafts is beneficial. Decompression of penis by escharotomy should be done for full thickness burns of the phallus when vascular compromise is suspected. Excision and grafting is done as soon as possible but efforts should be made to spare the dartos which has a rich venous network. If this can be preserved the problems of distal swelling, wound contraction and subsequent contracture can be eliminated. The laxity of penile skin enables about one fourth of the circumference to be excised and primarily closed. This can be a useful method when burns affect only the dorsum of the penis. Unmeshed grafts are preferable for better aesthetic appearance. Circumcision discarding non-burned penile skin is forbidden as it might be required for later reconstruction. Acute circumcision is indicated only for full thickness burns of the prepuce. When testes are exposed, they can be sutured together for stability and covered with partial thickness skin graft. Intrinsic contractures of penis, scrotum or vulva are rare but extrinsic contracture or webbing across the perineum and groin are more common.

Prevention of Contractures

To prevent contractures, lower limbs should be in abducted position during wound healing period with a spreading bar or wedge splinting. Active and passive exercises for hip joints, pressure garments with special inserts are also important in the prevention of contractures.

MANAGEMENT OF PERINEAL BURN CONTRACTURES/RECONSTRUCTION[8-11]

Gottlieb and Saunders[3] have classified burn scar deformities of perineum into quantitative and qualitative types.

Quantitative are those in which there is loss of skin surface and/or functional tissues, for example, most contractures. Qualitative scars are those which have altered skin texture, hypo/hyperpigmentation and changes in pliability. Although quantitative contractures are more disabling functionally, the reconstruction of qualitative ones is more difficult, a challenge that should not be neglected as it can lead to problems of sexuality. Perineal burn contractures have also been described as scar bands between the medial thighs crossing the area between genitalia and anus or a band extending from proximal medial thigh to perineum (Figs 28.7A to 28.8).

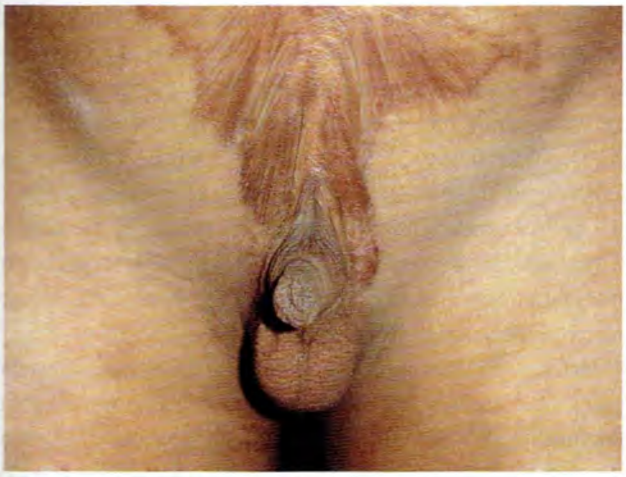

Fig. 28.7A: Post burn scarring of suprapubic area extending to root of penis causing dorsal contracture of penis

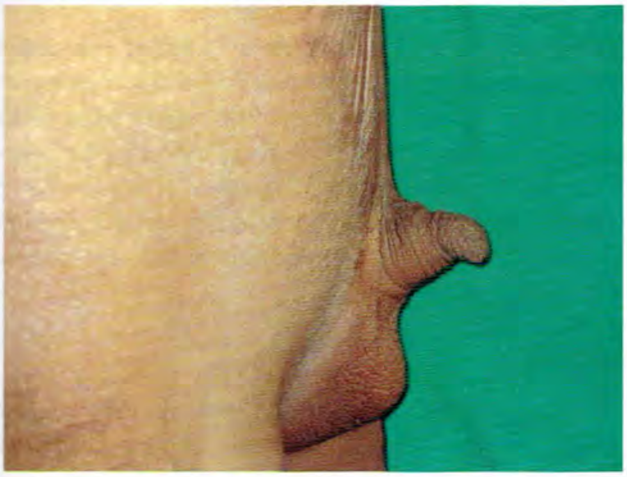

Fig. 28.7B: Same patient in lateral view showing the dorsal contracture of penis. Patient had difficulty during micturition as the stream of urine was directed horizontally. Patient required a dorsal release at the root of penis

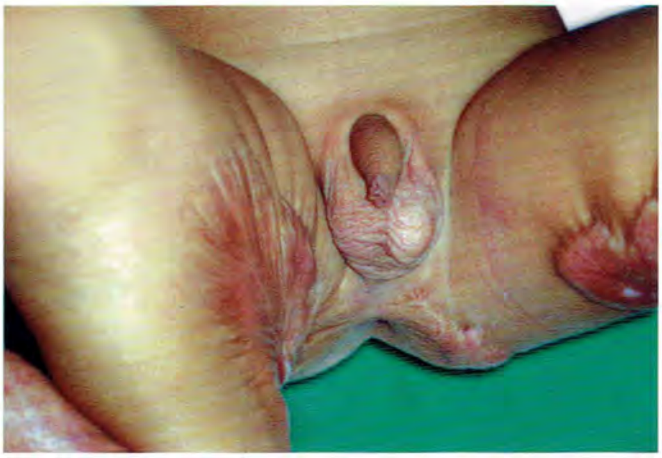

Fig. 28.8: Showing a child with post burn contracture of perineum

In spite of early surgical intervention and early mobilization perineal contractures occur in a small percentage of patients. These numbers are higher in places where education levels are low and patient follow up is poor. Small contractures can usually be treated with Z plasty or its variants. Scar excision and primary closure, local rotation and transposition flaps are other methods of dealing with small contractures. Larger contractures need to be released and grafted (Figs 28.9A and B). Grafting of both thighs leaving the intervening portion intact as advocated in double release or bipedicled release for elbow and popliteal fossa can be done. If lower abdomen is spared, this skin can be brought down to avoid skin grafting in the suprapubic area. It is a good method but is rarely applicable as in most cases of burns of genitalia and perineum which require surgery, the lower abdomen is also burnt. When the undesirable scar is not extensive and the adjacent skin is normal and adequate, tissue expansion is an option. It allows one to get qualitatively normal skin without any donor site deformity. However, expanders in the perineal area are fraught with dangers. High incidence of infection and exposure is seen. Patients find it very cumbersome to sit, move around and perform their normal daily activities with the bulges of the expanders. However, when the expanders are successful they produce the best results. Alternatively full thickness skin donor sites are expanded and used for better cosmetic results.

Postoperative rehabilitation includes massage, stretching and leg abduction exercises, maintenance of abducted position with spreading bar splint and regular usage of pressure garments for three to six months. Postoperative rehabilitation did not appear to influence recurrence which has been reported to be as high as 20%.

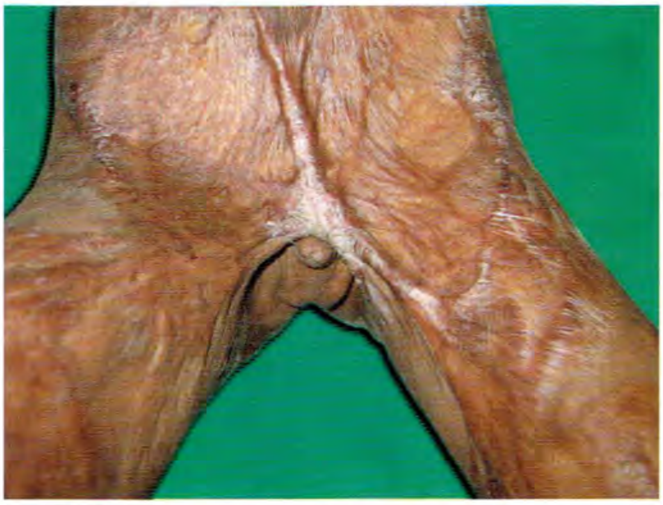

Fig. 28.9A: Severe post thermal burn contracture of perineum and groin causing the penis to bury beneath the contracted skin

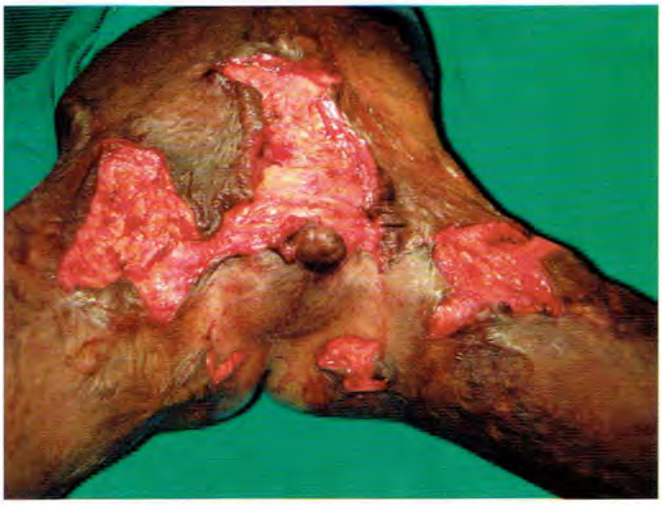

Fig. 28.9B: Same patient showing released contracture followed by skin grafting

Indications for surgery are decreased range of motion and leg abduction. The ideal time for contracture release is after the scar has matured fully. This can take from six months to two years. An exception to this rule is when patient needs relief from a severe functional disability.

Intrinsic contracture of penis results from injury and secondary scarring of dartos, Bucks fascia, tunica or corpora. Small scars limited to dartos or Bucks fascia can be treated with incision or excision. More severe deficiency needs excision followed by reconstruction with dermal graft and covered by Byar's or any other local flap. If there is scarring of urethral meatus dilatation may be enough for mild cases, Z-plasty and other interdigitating flaps are reserved for more severe cases.

Total reconstruction of penis was started with tube pedicle flaps which gave way to the axial pattern flaps, muscle flaps which have now been replaced by state of art neuro-sensory fasciocutaneous free flaps. The best results have been obtained by the neurosensory radial forearm flap which can offer both erogenous and protective sensation with prosthesis for erectile function. Other methods which have been used include bilateral gracilis muscle flap and rectus abdominis muscle flap. The girth provided by the flap should be adequate to contain both the neourethra and the prosthesis. Subtotal penile loss is treated the same way adding the flap to the preserved portion of corpora and urethra.

Vulvar injuries are rare but when they occur they might lead to webbing which compromises normal sexual activity and cause difficulty in micturition. If adjacent tissues are healthy and pliable, Z-plasty, local transposition flaps or V-Y advancement flaps can be done. Basically flaps from normal tissue are used to break up the scar contracture line. If surrounding skin is scarred, the contractures have to be released and split skin grafted.

Extensive burns involving buttocks can sometimes result in perianal stenosis or hooding which can impede fecal evacuation. These patients, on closer examination, may reveal entrapped unburned tissue. This can be released by incising the burnt tissue from perineal body to coccyx through the stenotic orifice and everting the unburned tissue away from the anus. Lateral excisions of bands of burnt skin allow recreation of gluteal folds.

Repeated discussions concerning realistic expectation regarding appearance, sexual function and reconstructive options is important. This should be started early in the acute phase and continued throughout the treatment period till reconstruction is complete. Discussions regarding nature of deformity, surgical improvement, various available options and realistic expectations of both the patient and surgeon are critical.

SUMMARY

- Burns involving perineum and genitalia only are rare. They are usually seen in patients who have sustained large percentage of burns more than 40%.
- Superficial burns are treated conservatively while deeper wounds need excision and resurfacing. Full thickness burns need reconstructive procedures.

- Urinary diversion with catheter is usually required but fecal diversion is not done.
- Perineal contractures are a rare complication.
- Indications for surgery include decreased leg abduction as well as urinary and bowel complications.
- Surgery for perineal contractures usually requires simple methods like local flaps and skin grafts and complicated procedures are reserved for reconstruction of genitalia.
- Local flaps are superior to skin grafts which should be used only when local tissues are unavailable.
- Aggressive rehabilitative techniques help preventing recurrences.

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Chapter 29

Pediatric Burns

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INTRODUCTION

In the pediatric age group, as is true for adults, a burn injury is the second most important cause of trauma-related deaths ranking after motor vehicular accidents only. Besides causing death, a large number of children survive burns only to have lifelong disfigurement, loss of function and physical deformity leading to psychological sequelae. A burn injury not only leaves the scars on the skin but also on the mind of the child.

Burnt children account for almost 15 percent of all burn patients admitted to a large burn center. The management of a burn patient is complex and requires a multidisciplinary approach. When the patient happens to be a child, the challenges are further magnified and a competent and unified approach to patient care becomes mandatory.

MODE OF INJURY

Most of the burns in children occur in the child's own house. The age of the child and his environment suggest subgroups of children at risk for burn injury.[1] The infants and toddlers less than 2 years of age are frequently burnt. An important factor leading to burns even in neonates is overcrowding in a small house, where a small area in the room serves as kitchen (with floor level cooking). The victim is looked after by his/her siblings, who are themselves no more than 5-6 years old when the mother is away at work to earn. Infants and small children are also the only victims when clusters of 'jhuggis' (huts) catch fire and they are either sleeping alone or unable to rescue themselves (Figs 29.1A and B).

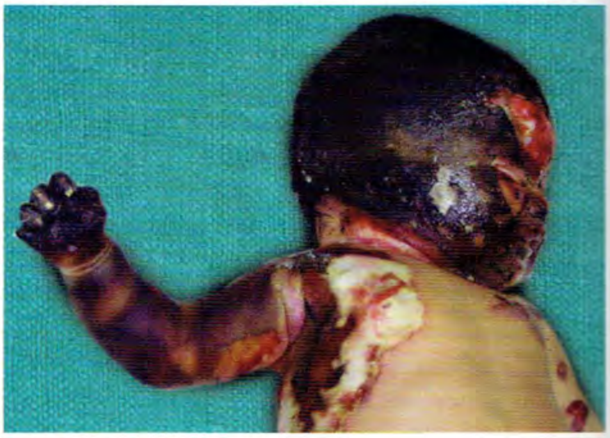

Fig. 29.1A: A 3-month-old infant with 35% full thickness burns sustained accidentally in a 'jhuggi' fire when child was left unattended by mother

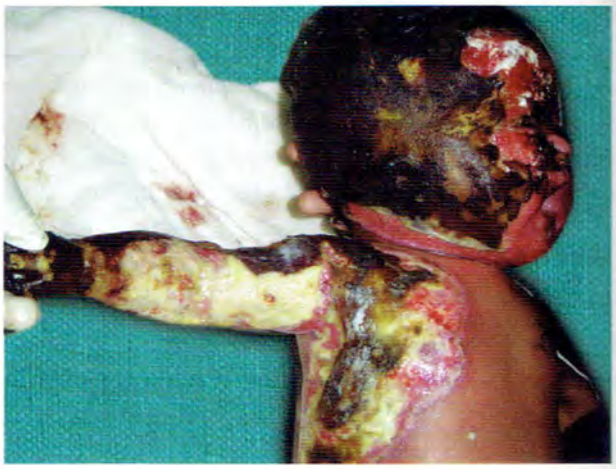

Fig. 29.1B: 2 weeks post burn picture of same patient showing eschar separating and wounds starting to granulate from underneath

Moreover, children out of curiosity actively search and manipulate their physical environment. Scald burns are the commonest type of thermal injury in children. They usually suffer scalds by pulling down or knocking hot liquids on to themselves. Immersion scalds can occur when a child steps into a tub of hot water or stumbles onto a container of hot milk or cooked food (Figs 29.2 and 29.3). Children also feel a temptation to experiment with match boxes. Fireworks during Diwali and other festivals is a major cause of burns in children. In winter season, it is a common practice for adults in our country to collect wooden sticks and old worn-out automobile tyres and ignite them to keep warm. Children often imitate adults and get burnt by this practice.

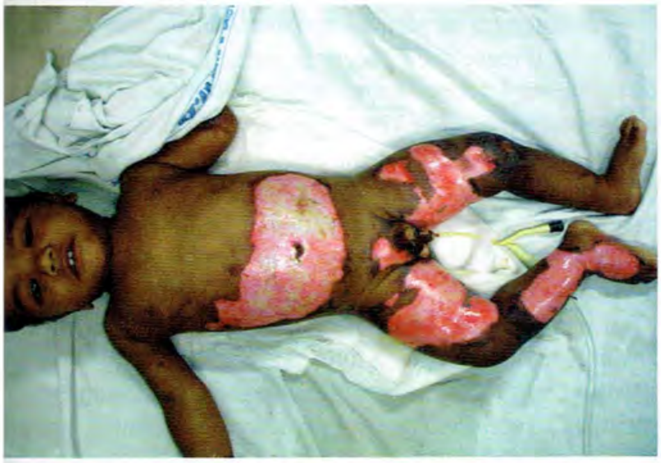

Fig. 29.2: A 1½ year-old child with 20% scalds due to child stumbling into a container of hot milk

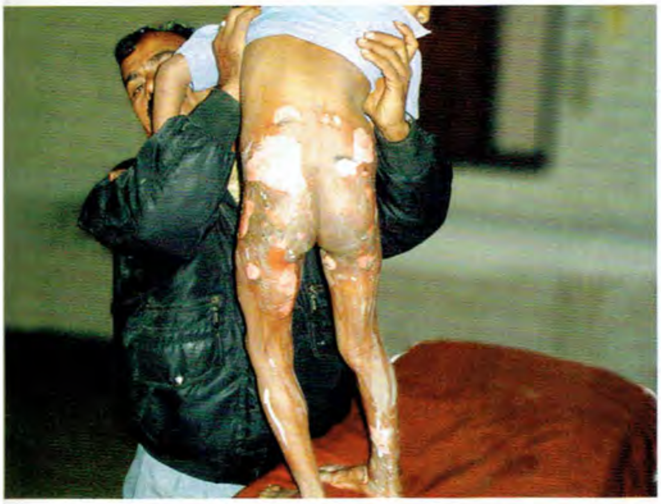

Fig. 29.3: Another child with 20% superficial dermal scalds due to hot water bucket falling on him

Electrical burns in children are generally caused by domestic voltage electric current which is 220-250 volts in India. Typically, a child bites an electric cord sustaining a disfiguring third degree burn of the lips[2] (Fig. 29.4). He may introduce a finger or an object such as a metallic hair pin into an electric socket and suffer an electrical injury. Chemical burns may be caused by strong acids and alkalis used for cleaning household toilets and drain and quite commonly stored carelessly.

Majority of burn injuries in children are accidental. Perineal scalds (Fig. 29.5) and acid burns are very common in children. A very small percentage of pediatric burns are due to homicide and child abuse in India, although they are frequently reported from developed countries (Fig. 29.6).

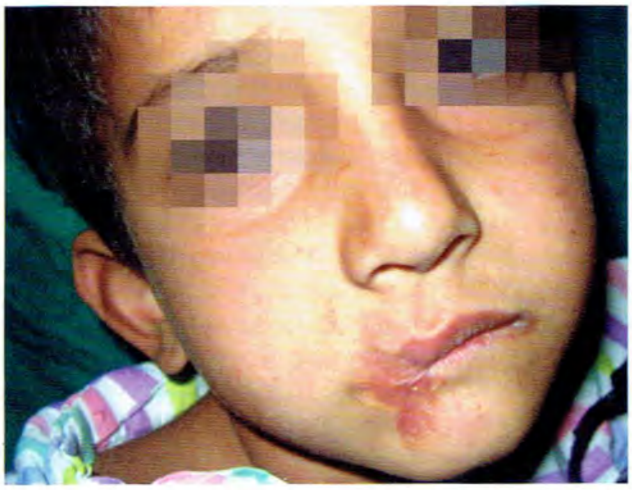

Fig. 29.4: Post burn microstomia following electric burn sustained by a 4-year-old child while chewing on an electric cord

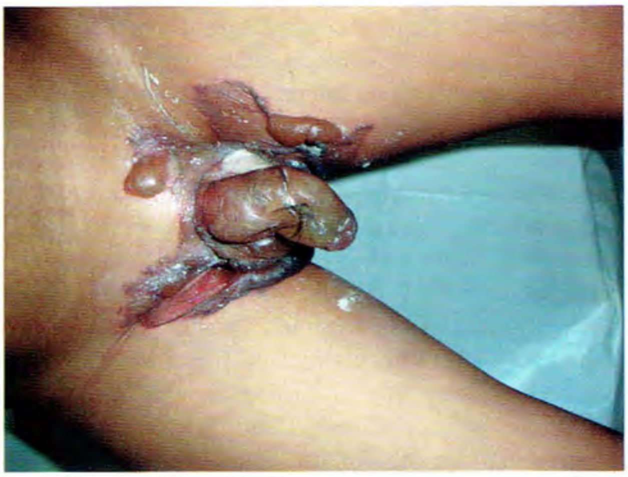

Fig. 29.5: Perineal scalds because of spilling of hot tea from a cup lying on the table, when child pulled down the tablecloth

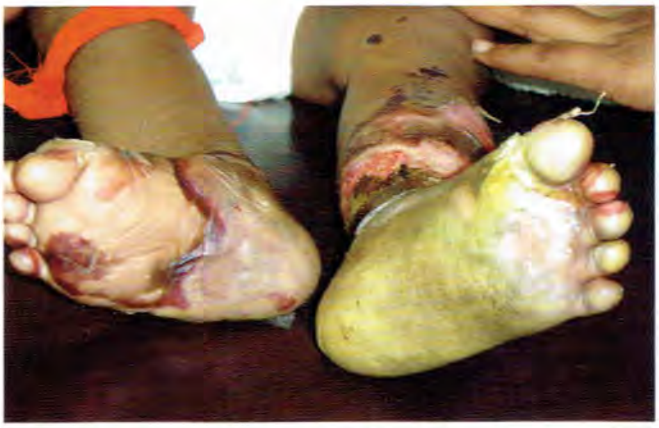

Fig. 29.6: Case of child abuse with full thickness burn both feet (soles) after his feet were forcefully dipped into hot water

PATHOPHYSIOLOGY

A child is not merely a small adult. Children react differently from adults to a thermal trauma, both in their local reaction and systemic response. Therefore, in children, especially those less than 2 years old, there are certain special physiologic considerations which have to be kept in mind while managing burns. One of the most important feature of infants is their larger surface area relative to weight when compared with older children and adults. The evaporative water losses and hence the heat loss is greater for children compared to adults because of their larger surface area. The temperature regulatory mechanism in children is also different in different age groups. For infants less than 6 months of age, the body temperature is usually regulated by non-shivering thermogenic response wherein the stores of brown fat are catabolized under the influence of norepinephrine. Since this process utilizes large amounts of oxygen, prolonged hypothermia can lead to excess lactate production leading to acidosis. After 6 months of age, children are able to shiver but body heat is still lost as their surface area per unit body weight is larger than that of adults. To reduce this loss external sources to preserve body temperature are required. Therefore, infants and children with burns have to be treated in cubicles where the ambient temperature can be kept high so as to reduce the radiant and evaporative heat loss from the body to the environment.

The physical immaturity and smaller size of infants compared to adults result in significant structural and functional differences in cardiovascular system and maintenance of fluid balance. These differences affect the response of infant and child to hypovolemia and in turn, affect the management of fluid resuscitation following burns. Infants, in particular, rely heavily on their heart rate to increase their cardiac output and rates as high as 220 beats per minute are seen in hypovolemic infants following burns. This compensatory mechanism is easily exhausted making infants more vulnerable to burn shock.

The infants also have less mature renal function as compared to adults. The glomerular filtration rate and renal blood flow of an infant is lower than that in adults. In fact the glomerular filtration rate in infants reaches adult levels only after approximately 9 to 12 months of age because of delayed maturation of glomerular and tubular function. During this time, the infant has approximately half the osmolar concentrating capacity of the adult and water load is handled inefficiently. During the first several weeks of life the infant is likely to retain a large portion of water load administered as part of burn resuscitation. This may apply to a slightly lesser degree to other substances such as sodium. All these differences are accentuated by the stress of burn injury. The syndrome of inappropriate anti-diuretic hormone secretion (SIADH) is associated with many acute illnesses including burns. The elevated ADH levels result in conservation of water and high sodium excretion causing hyponatremia and water overload. The fluid overload in children can also precipitate convulsions due to dilutional hyponatremia.

It is important to know that normal heart rates of children are higher than that of adults (Table 29.1).This helps in meeting the substantially higher metabolic requirements of an infant. The blood pressure is also on the lower side in infants and children and it increases with age because of corresponding increase in the systemic vascular resistance.

Table 29.1: Normal heart rate and Blood pressure in children

Age group	*Heart rate (per minute)*	*Blood pressure (systolic/diastolic)*
Newborn	140	65/45 mm Hg
1 year	110	75/50 mm Hg
3 years	100	90/60 mm Hg
8 years	90	95/65 mm Hg
10 years	80	100/70 mm Hg

FIRST AID

First aid is carried out by parents, other relatives or any other person present at the site of accident, most commonly the house of the child or neighbor. The public must be made aware of how to prevent burn injuries in children and what measures should be taken in case the unfortunate event takes place. Media, especially television, has a great role to play where the invited burn specialists teach the public about it.

1. The burned area is cooled immediately with tap water (preferably running water) to bring the burning skin to normal body temperature. Very cold water or ice should not be used as this can lead to hypothermia.
2. Rings, bracelet or watches, etc. should be removed from the affected area as they may cause tightness or constriction, when edema occurs.
3. After cooling, remove the clothing from the burnt area. However, do not try to pull out clothing that is stuck to the skin. This may cause more harm.
4. A cold compress such as a towel soaked in cold water may be soothing over the burnt area. This can be applied after the initial cooling.
5. Before going to hospital, cover the burned area with a clean sheet to prevent contamination and hypothermia.
6. Paracetamol or ibuprofen syrup may be given to decrease pain.
7. No topical agent should be applied to the burnt area lest the evaluation of depth of burns become difficult.

MANAGEMENT

Before definitive wound care commences, a protocol for rapid assessment of the injury and resuscitative measures to be taken is formulated.

Assessment of Burn Injury

Treatment priorities begin with the basics; Airway, Breathing and Circulation. To make a plan for further management, the burn injury should be assessed for:

- Percentage of total body surface area involved
- Depth of burn injury
- Location of the burns
- Any associated injuries

Estimation of Burn Size

1. **Lund and Browder's Chart:** The percentage of body surface area involved can be readily assessed by standard Lund and Browder chart[3] (Table 29.2) which is an accurate technique for assessing percentage of burn surface area in children.

   In children, the head is proportionately a larger contributor to total body surface area (TBSA), while the lower limbs contribute less as compared to adults (Table 29.2). The head and neck comprises 19% of total surface area in an infant. With each year the surface area decreases by 1% till at 10 years of age it is similar to that of an adult. Similarly, each lower limb constitutes 13% of surface area of body in an infant and it increases by 0.5% per year till 10 years of age when it almost matches that of an adult (18%).[4]

   Another simple formula that can correct for differences in body surface area in children is, to subtract child's age in years from 12 and the resulting number is added to the figure for adult head and neck or subtracted from the figure for both lower limbs.

   Example: At 8 years the head and neck constitutes 9 + (12 – 8) = 13% of total body surface area.

Table 29.2: Lund and Browder's chart showing changes in body surface area (%) with age

Area	*Birth to1 year*	*1 to 4 years*	*5 to 9 years*	*10 to 14 years*	*15 years*	*Adult*
Head	19	17	13	11	9	7
Neck	2	2	2	2	2	2
Anterior trunk	13	13	13	13	13	13
Posterior trunk	13	13	13	13	13	13
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5
Genitalia	1	1	1	1	1	1
Right upper arm	4	4	4	4	4	4
Left upper arm	4	4	4	4	4	4
Right lower arm	3	3	3	3	3	3
Left lower arm	3	3	3	3	3	3
Right hand	2.5	2.5	2.5	2.5	2.5	2.5
Left hand	2.5	2.5	2.5	2.5	2.5	2.5
Right thigh	5.5	6.5	8	8.5	9	9.5
Left thigh	5.5	6.5	8	8.5	9	9.5
Right leg	5	5	5.5	6	6.5	7
Left leg	5	5	5.5	6	6.5	7
Right foot	3.5	3.5	3.5	3.5	3.5	3.5
Left foot	3.5	3.5	3.5	3.5	3.5	3.5
Total:	100	100	100	100	100	100

At 8 years both lower limbs constitutes 36 – (12 – 8) = 32% of total body surface area i.e. 16% for each lower limb.

2. **Wallace's Rule of 'nine'**: In children over 12 years, burn wounds can be estimated by this formula which is also used in adults.[5]
   According to this,
   - Head and neck – 9% TBSA (9 × 1)
   - Each upper limb – 9% TBSA (9 × 2)
   - Anterior trunk– 18% TBSA (9 × 2)
   - Posterior trunk – 18% TBSA (9 × 2)
   - Each lower limb– 18% TBSA (9 × 2 × 2)
   - Perineum and external genitalia – 1% TBSA.
   - Total = 100%
3. **Rule of 'five':** Lynch and Blocker developed a formula for estimating the extent of burns in children.[6] It works very well in infants where head and neck, anterior and posterior aspects of trunk are 20% each and each limb constitutes 10%. For older children, this is slightly modified. The head, posterior trunk and lower limbs are 15% each, anterior trunk is 20% and each upper limb is 10%.
4. **Rule of Palm or Hand:** For all age groups, palmar surface of one's hand represents his/her 1% TBSA. This is useful for assessing scattered burnt areas. Only second-degree burns or greater should be included in the TBSA burnt for burns management.

Estimation of Burn Depth (Figs 29.7 to 29.10)

The burn depth estimation is discussed in detail elsewhere in this volume. During infancy, the epidermis is loosely connected to the dermis. As a result, blisters form more readily in burns in infants. The stratum corneum layer of epidermis is thinner in children so that scalds cause deeper burns in children compared to adults. The amount of sebum and sweat production in infant's skin is also less than that in older children and adults. Due to all these reasons, a child's skin is susceptible to greater damage by heat and chemicals even if exposure is for short duration.

Many burn wounds which initially appear superficial may eventually turn out to be deeper and may require skin grafting. A scald injury which looks bright red and is taken as a superficial burn in an adult may actually turn out to be full thickness burn in young children.

A severe burn is one that covers greater than 15% of the body surface area or involves the face or perineum. Second degree and third degree burns of the hands or feet, as well as circumferential burns of the extremities are also classified as severe. Inhalation injuries resulting in bronchospasm and impaired pulmonary function must be considered as severe burns.

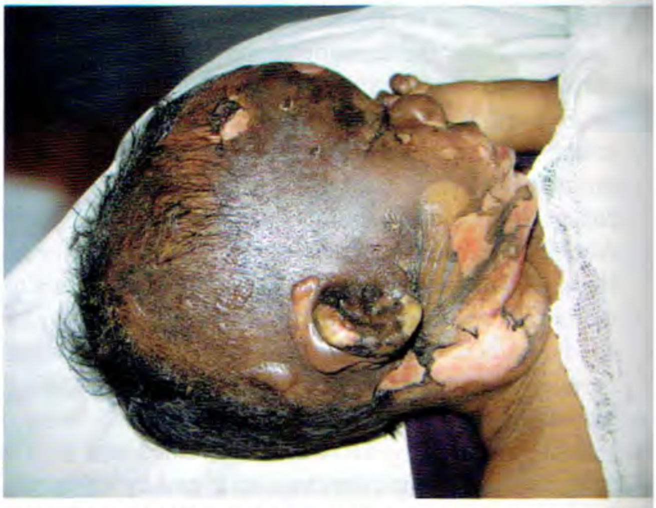

Fig. 29.7: Accidental superficial dermal burns of face in an infant

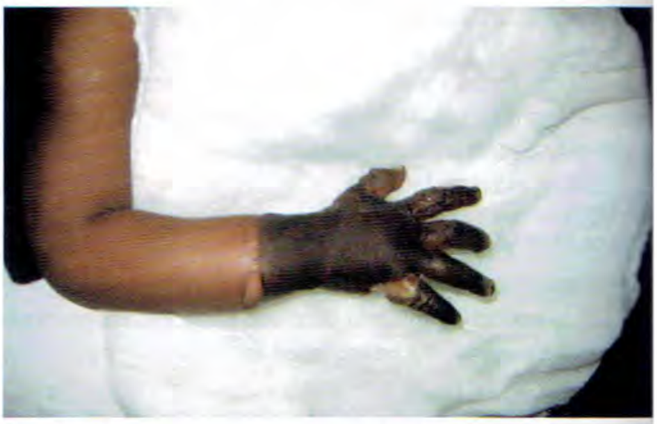

Fig. 29.8: Full thickness burns of hand and distal forearm of a child

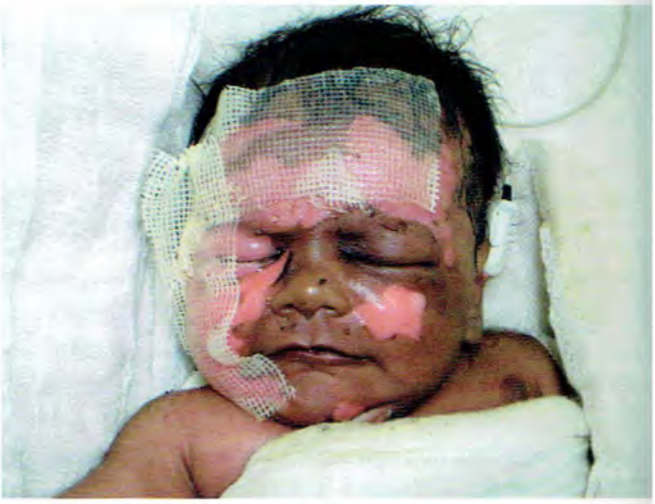

Fig. 29.9: Superficial dermal or superficial second degree scalds of face of an infant

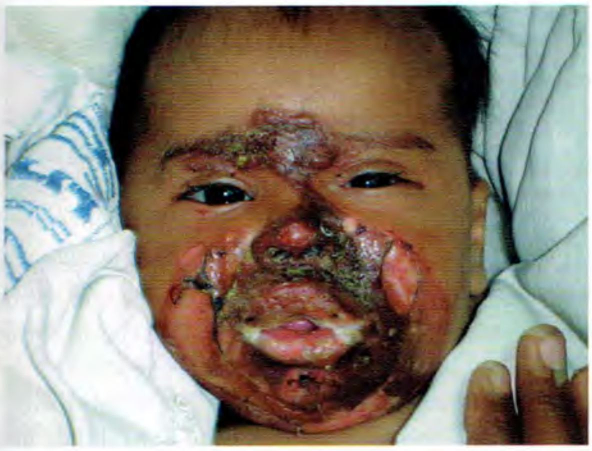

Fig. 29.10: Another infant with deep dermal or deep second degree burns of face

Indications for Admission to Hospital

The indications to admit a child to a burn care facility are:[7]

1. Extent of the burn – Children with more than 10% burns.
2. Areas involved – Involvement of face, neck, both hands, both feet or perineum.
3. Types of burn – Children with electrical or chemical burns, third degree burns of greater than 5%.
4. Associated injuries – If burns are associated with soft tissue trauma or head injury, fractures etc.
5. Inhalation burns.
6. Social problems – If there is suspected child abuse or neglect, self-inflicted burns, poor guardianship at home regarding wound care or psychological problems present, ambulatory management may not desirable.

Initial Management

Although most children are victims of scalds, the initial management must give first priority to airway. If a child is involved in a fire in closed space or "Jhuggi" (hut) fire, inhalational injury should be strongly suspected.The upper airway is susceptible to burn injury, whereas the subglottic space appears protected. Facial burns, singed nasal hairs or eyebrows and acute inflammation of the oropharynx suggest inhalation injury. Smoke inhalation may be associated with carbon monoxide toxicity; 100% humidified oxygen should be given if hypoxia or inhalation injury is suspected. A carboxyhemoglobin assessment should be performed for any suspected inhalation exposure (a house or closed space fire or a burn victim who requires CPR). Hoarseness on vocalization is also consistent with supraglottic injury. Some children with inhalation injuries require intubation or tracheostomy and mechanical ventilation (Figs 29.11A and B).

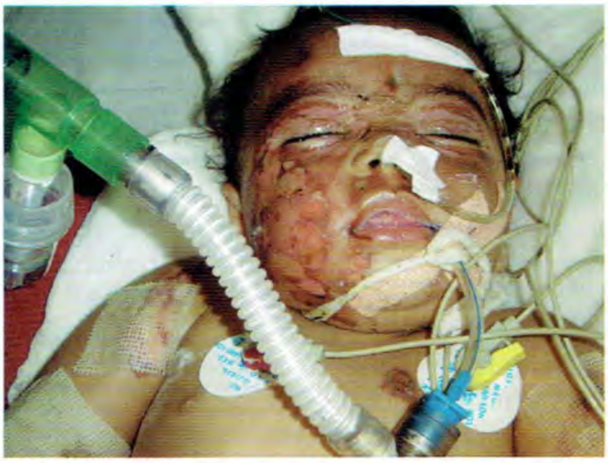

Fig. 29.11A: Patient intubated for mechanical ventilation even though child sustained only 10% scald with hot tea following which parents poured bucket full of water on his face and child aspirated hot tea and water both leading to aspiration pneumonitis

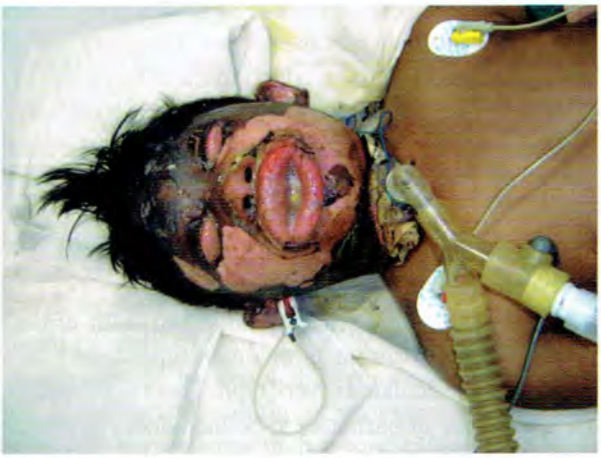

Fig. 29.11B: Another child with 10% facial burns with smoke inhalation injury requiring tracheostomy for ventilation

Fluid Management

Adequate fluid resuscitation in the first 24 hours post burn is of utmost importance. Many fluid resuscitation regimes have been advocated over the last 50 years. Pruitt et al[8] recommended 3 ml/kg/% burn surface area as resuscitation formula for thermally injured children. Baxter reported 4 ml/kg/% BSA for fluid calculation[9] while Warden et al[10] recommended a mean fluid requirement of 5.8 ml/kg/% BSA for children.

Compared to adults, children require fluid resuscitation for burns as small as 10-20 % BSA because of their limited physiological reserve. Various studies have demonstrated that children with burns less than 30% area require less fluid/kg /% burn than those with more than 30% burns.[11,12]

There are many popularly used formulae for intravenous fluid therapy. Though all the formulae are quantitative and mathematical to a certain extent, clinical judgment is very essential while deciding the quantity and quality of fluid given to children. No single formula can be adhered to strictly in the management of a burnt child. The most important fact underlying this decision is not to overload the vascular system and at the same time not to under-resuscitate the child. In Parkland / Baxter formula, the total fluid requirement is estimated on the basis of extent of burn and weight (in kg) of patient only, without any additional daily maintenance fluid. The fluid calculated as per these formulae in infants especially with smaller burns may not suffice as it may not even meet the total daily requirement. For example, in a 10 kg child with 20 % burns the daily fluid requirement is 1000 ml. However, the amount of resuscitation fluid calculated for the same using Parkland formula (4 × 20 × 10) comes to only 800 ml in first 24 hours. This results in gross under-resuscitation of the patient. Therefore, the daily maintenance fluid has to be added to these formulae in the form of dextrose saline. This is given as N/2 saline with 2.5 % dextrose or N/4 saline with 3.75 % dextrose according to the age of the child (For less than 2 months of age N/5 or N/6; 2 months to 5 years, N/4 and for 5-12 years, N/3 or N/2). The addition of dextrose decreases the incidence of hypoglycemia in infants in the first 24 hours.

On the other hand, in extensive burns in children, Parkland/Baxter formula is again unsatisfactory as the amount of fluid calculated is very high. There is no restriction to the percentage of burn area to be included. For example, a 10 kg child with 80% burns would be predicted to require 10 × 80 × 4 = 3200 ml fluid in first 24 hours although the daily fluid requirement for the same patient is only 1000 ml. This large amount of ringer lactate, if given to the child, can result in fluid overload and resultant hyponatremia can precipitate convulsions in children. Therefore, a formula with a ceiling of 50% TBSA is essential along with addition of daily fluid requirement. For this purpose modified Brooke's formula satisfies all the requirements and is followed in our burn unit as well.

According to modified Brooke's formula, the first 24 hours requirement is calculated as follows:

2 ml × Wt in kg × % BSA burnt + Daily maintenance

The fluid of choice is crystalloid, namely ringer lactate. Daily maintenance fluid is given in the form of N/2 dextrose saline (0.45% NS with 2.5% dextrose) or N/4 dextrose saline (0.23% NS with 3.75% dextrose) and is calculated as per the weight of the child (Table 29.3).

Table 29.3: Daily maintenance therapy in children

Weight of child	*Daily maintenance fluid (ml/kg /day)*
0-10 kg	100 ml/kg
10-20 kg	1000 ml + 50 ml/kg for every 1 kg above 10 kg
More than 20 kg	1500 ml + 20 ml/kg for every 1 kg above 20 kg

Half of this fluid is given in first 8 hours and second half in next 16 hours. In the second 24 hours, the formula recommended for use is 1ml/kg/percent BSA burnt of ringer lactate + Daily maintenance (same as above).

The decision to use colloids or crystalloids is controversial. The rationale for giving crystalloids in the form of ringer lactate and avoiding colloids in the first 24 hours is that following burns it is the plasma which is lost from the intravascular compartment into extravascular space because of increased capillary permeability. Since ringer lactate is equivalent to plasma without proteins, it restores the intravascular pressure without adding to the loss of proteins into extravascular space. Colloids on the other hand, add to the loss of high molecular proteins from vascular compartment to extravascular space till such time that the capillary permeability is increased, usually between 18 to 24 hours. By 48 hours post burn, even though the permeability is restored to normal, the leaked out proteins with their water content cannot be reabsorbed back into the circulation easily. This increases the chances of pulmonary edema and peripheral edema persisting for a very long time with deleterious effects on the glomerular filtration, coagulation and cardiac function.[13,14] In studies by Baxter, it has been shown that colloids probably do not exert any measurable expansion of plasma over and above that exerted by ringer lactate till second post burn day.[9]

Martin et al compared patients receiving only colloid based fluids in first 24 hours and those receiving a combination of colloids and crystalloids and reported that the only colloid regime used much less fluid during resuscitation with much reduced rates of ARDS, hypoalbuminemia and sodium disturbances but more peripheral and pulmonary edema and renal impairment.[15]

So, the general consensus is that in first 24 hours, only if the fluid requirement is very high as in extensive pediatric burns, colloids can be added to crystalloids in the regime. In massive burns if only large volumes of crystalloids are given, the intravascular volume is expanded but the plasma oncotic pressures is not restored as they are devoid of proteins. On the other hand, the plasma oncotic pressure is restored faster with colloids because they contain high molecular weight molecules. Crystalloids in large amounts dilute the remaining proteins further in the intravascular space thus decreasing the oncotic pressure as well. As a certain level of oncotic pressure is essential to maintain glomerular filtration (GF), any further decrease in this pressure will affect the GF and thus decrease the urine output, which should be actually increasing. This gives a false impression that fluid resuscitation is inadequate and by fault more crystalloids may be infused to increase urine output, resulting in volume overload without improving the oncotic pressure. In such circumstances, infusion of colloids is required to improve plasma oncotic pressure and increase GF.

In the second 24 hours post burn, many centers prefer to add colloids to their fluid regime.[15, 16] In our unit we use only crystalloids. **Colloids are added only if the plasma oncotic pressure is very low, indicated by low urine output, despite adequate fluid replacement by crystalloids in first 24 hours.** Our belief is that the capillary permeability is restored to normal only by 48 hours and giving colloids prior to this stage will increase the peripheral tissue edema and pulmonary edema which will persist for a longer time as these high molecular weight proteins are not reabsorbed back. Colloids are given in the form of 5% albumin or plasma in a volume of 0.3-0.5 ml/kg/ % BSA burnt.

Since the routine formulae are exclusively weight-based and in children there is no linear relationship between weight and surface area, there are problems of under and over resuscitation in pediatric age group. To overcome this, studies have shown the benefits of using Galveston formula based on body surface area (BSA) calculated from the height and weight of the child from standard nomograms.[17,18] According to this formula, the amount of fluid administered in first 24 hours is calculated as 5000 ml/m^2 BSA burn + 2000 ml/ m^2 BSA, with half the volume given in first 8 hours and second half in next 16 hours. This has not yet been standardized and is not being used worldwide.

Monitoring of Fluid Resuscitation

The best way to monitor the adequacy of fluid resuscitation is by measuring pulmonary capillary wedge pressure. Since this is not routinely practicable in a clinical setting, hourly urine output measurement is the most practical and easily done to know the adequacy of resuscitation. The physiologic goal is maintaining urine output of at least 1ml/kg body weight/hour. During first 48 hours, adequate urine output is considered as follows (Table 29.4).

Table 29.4: Required urine output according to age

Age of Child	*Urine Output*
0-2 years	10-20 ml/hour
2-5 years	20-30 ml/hour
Above 5 years	30-50 ml/ hour

The child's pulse, respiratory rate and blood pressure are also monitored closely. A close watch is maintained on serum electrolyte levels because rapid fluid and electrolyte shifts in burnt children can result in cerebral edema and convulsions. Another parameter which has to be monitored for assessing adequacy of fluid resuscitation is the hematocrit which varies in children according to age. Both the hemoglobin and hematocrit are high at birth and then decrease in a few days. At one year, hematocrit is 35 % which gradually increases to 40% at 12 years. The fluid infusion is regulated to maintain hematocrit values between 35 and 40%.

From third day onwards, as the child is gradually started on oral feeds, intravenous fluids are gradually reduced and omitted. Once the capillary permeability is restored, urinary volumes far exceed the total of oral and intravenous intake due to return of fluid from the extracellular compartment into the intravascular compartment.

Children with partial thickness burns that cover less than 15% of the body surface may be given oral fluids in the form of milk or other clear liquids, but carbonated beverages should be avoided as they may contribute to gastric dilatation and vomiting. Under most circumstances, children with burn injuries in excess of 15% of BSA should have a secure intravenous catheter placed, either percutaneously or via a cut down. Unburned sites should be used for catheter placement, whenever possible.

Adjunctive Management

- **Nasogastric tube** – To prevent acute gastric dilatation, vomiting or aspiration, a nasogastric tube is inserted and the stomach is emptied. This is recommended for all children with burns of 20% or more of body surface area.
- **Urinary catheter** – It is routinely inserted in all children with > 20% burns to monitor hourly urine output.
- **H_2 receptor antagonists** – Children with burns requiring admission should be given H_2 receptor antagonists (e.g. Ranitidine) and antacids for prophylaxis against gastroduodenal erosions and ulcerations (Curling ulcers).

- **Tetanus prophylaxis –** Tetanus toxoid is administered for prophylaxis against tetanus along standard recommended lines. With the great emphasis by the Government of India in immunizing children against many diseases in the first year of life, tetanus being one of them, many children may be already immunized at the time of burn. In children not properly immunized, passive protection is also provided with 250 units of tetanus immunoglobulin.
- **Analgesia and sedation –** Pain relief and sedation must be preceded by correction of hypovolemia and hypoxia as they are the cause of restlessness in a burnt patient. Morphine (0.05-0.1 mg/kg IV every 2-4 hours), pethidine/pentazocine and promethazine combination in appropriate doses are used intravenously (slowly) on 'as and when required' basis. After the initial shock period is over, they may be given intramuscularly. For children with minor burns being treated on outpatient basis, Paracetamol (10-15 mg/kg every 6 hours) alone or its combination with ibuprofen, nimesulide can be used.
- **Blood transfusion** – These are not required in the first 48 hours unless the child is anemic. It may be required later to maintain hematocrit between 35 and 40% and a hemoglobin level of more than 10 g/dl (Fig. 29.12).

Systemic Antibiotics

Burn wounds provide large, warm, moist and protein-rich medium for growth of microorganisms from endogenous and exogenous sources. Children with burns are also more susceptible to infection because of depressed immune system. Consequently sepsis is the leading cause of death from burns. Routine use of prophylactic systemic antibiotics, however, is not recommended as it leads to rapid emergence of resistant strains. For protection against beta hemolytic streptococcal infections especially in children, it was earlier recommended that crystalline penicillin should be used prophylactically for first 5 days following burns. However, with changing wound flora and with emergence of new strains even this practice has been discontinued at many centers. Prophylactic penicillin therapy is indicated during the phase of autogenous skin grafting since streptococcal infection is likely to occur at that time.

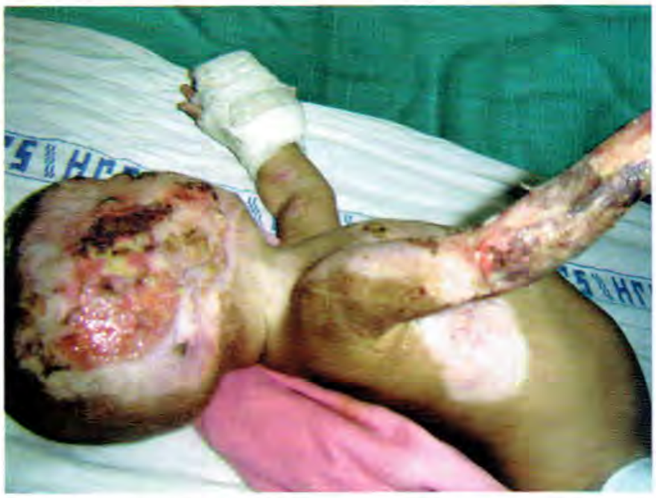

Fig. 29.12: Infant with 20% post burn raw area with severe anemia. Wounds show unhealthy pale granulation tissue. Requires building up with blood transfusion and nutrition till wounds are prepared for grafting

Routine bacteriological monitoring of the burn wound is carried out with surface swab cultures. Burn wound biopsies have been used to provide a quantitative estimation of the bacteria in the wound and infection is diagnosed when more than 10^5 bacteria are present per gram of tissue or there is invasion of subjacent healthy tissue. Local signs of burn wound sepsis include development of black or purple necrotic areas in the wound and hemorrhage in the sub-eschar fat. Bacteria generally isolated in the burn wounds are *Pseudomonas*, *Klebsiella*, *Staph. aureus*, *Streptococcus faecalis*, *Proteus* and *E.coli*.

Whereas no clinical sign is diagnostic of septicemia, it is clinically suspected with a change in condition of the wound, presence of fever or hypothermia, altered sensorium, tachycardia, tachypnea, abdominal distension, diarrhea and oliguria. Although infection is accompanied by fever and leukocytosis, sepsis in burnt children may manifest with hypothermia and leucopenia.

For burn sepsis, systemic antibiotics are the mainstay of treatment (Figs 29.13 and 29.14). If the culture and sensitivity reports are available the appropriate antibiotic treatment is directed towards the identified bacteria. Otherwise, antibiotics are started in broad spectrum combinations (e.g. cephalosporin and aminoglycosides) empirically, pending culture reports. Antibiotics are started at the first sign of sepsis, in maximal therapeutic doses and stopped only after seven days or so.[19]

Nutrition

Nutrition plays an integral part in the treatment of a pediatric burn patient. Adequate energy and protein intake is required to meet the increased metabolic demands of the hypermetabolic state of the burn injury as well as normal growth and development. Prompt initiation of nutritional

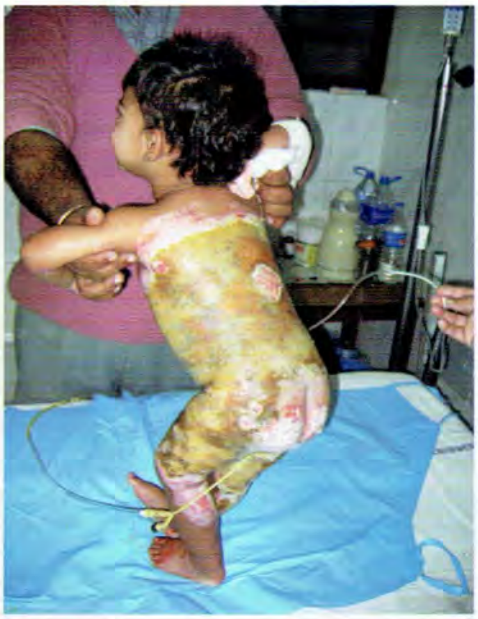

Fig. 29.13: A 2-year-old child with burn wound sepsis due to *Pseudomonas*

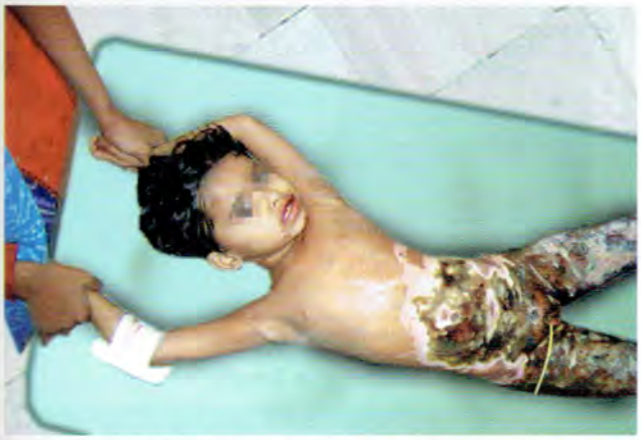

Fig. 29.14: Child with typical gram-negative sepsis

support is crucial and is associated with reduction in cost of hospitalization.

Enteral route is preferred for nutritional support and is generally available. Oral feeding is instituted by third post burn day and by the fifth day it is increased to meet the predicted caloric requirements. If the child is unable to take orally, a nasogastric tube may be placed, through which freshly prepared feeds, at body temperature, can be given as drip. The feeds should have 1 kcal/ml and a low osmolarity (300-700 mOsm/l) or else they lead to osmotic diarrhea. Parenteral alimentation or supplements are necessary if the child is having vomiting, diarrhea or malabsorption. Parenteral alimentation is done with 10-20% dextrose solution with emulsified fats and amino acid solutions.

The daily caloric requirements in children can be estimated by the following formulae:

1. Curreri's formula:[20] 60 kcal/kg body weight + 35 kcal/percent burn
2. Carjaval's formula:[21] 2200 kcal/m^2 of burnt area + 1800 kcal/m^2 BSA

Along with calories, an adequate amount of protein is given which is important to improve resistance and survival. One of the formulas suggested for estimating protein requirements in a burnt patient is 3 gm/kg + 1 g for every one percent of BSA burnt. No more than 4 gm/kg protein should be provided. Fats are concentrated source of energy but excessive intake of fat has been associated with many complications. 5-15% of non-protein calories should be provided as fats and rest as carbohydrates. Dietary supplements of vitamins and trace elements including zinc, copper, chromium and molybdenum are also important for wound healing and given orally or in intravenous fluids.

Management of the Burn Wound

Burn wounds in children are best treated by dressings as the child will be able to move around and play more easily after he is out of the resuscitation phase. Perineal burns are a difficult area to treat in children. For this paraffin gauze dressing with topical antimicrobial cream can be applied directly on the nappy and the nappy can be changed as frequently as required.

Microorganisms proliferate rapidly in burn wounds, especially in those severe enough to impair immune function. Topical antimicrobials have an important role to play. They delay the interval between injury and colonization and maintain low levels of the wound flora. Three agents have proven efficacy for major burns viz., 1% silver sulfadiazine cream, 0.5% silver nitrate solution and11.1% mafenide acetate cream. Silver sulfadiazine [22] is most widely used. Leucopenia is common during first week of treatment due to marrow toxicity but usually resolves spontaneously.

Shallow burns are treated with daily dressings and local wound care until epithelialization occurs. They can also be treated by covering the denuded wound with skin allograft or xenograft or synthetic membranes available commercially. Collagen dressings have been found to be very helpful in superficial burns as they decrease pain and evaporative losses, seal the wound from the environment, makes the child more comfortable and encourages early healing (Figs 29.15A to 29.16C).

In case of full thickness circumferential burns or electrical burns where the distal vascularity is getting compromised, fasciotomy incisions should be given to improve circulation to the distal parts of the extremities (Figs 29.17A to C).

Children with deep dermal burns are particularly benefitted by early excision and grafting as it reduces morbidity, allows early recovery, reduces length of hospital stay, number of dressings and thus lessens the psychological trauma at such a young age. Blood should be kept ready in sufficient amounts for transfusion during surgery as children tolerate only small blood losses. A study on blood loss during excision in pediatric burn patients has shown loss of 2% of circulating blood volume for each 1% area excised and grafted.[23] Another important fact to be kept in mind during excision in pediatric age group is that the dermis is very thin. A true tangential excision leaving behind viable dermis can be difficult at times and may actually turn out to be excision till subcutaneous fat leading to marked cosmetic deformities later on. This is also to be remembered while procuring graft from the donor sites especially if the donor site has to be reused.

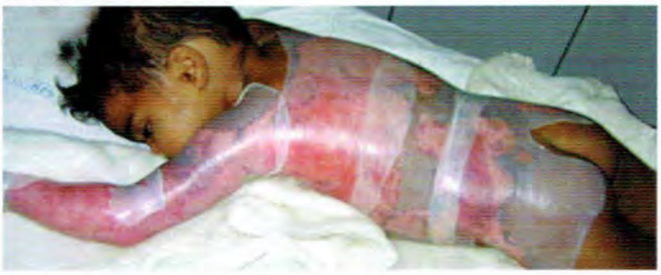

Fig. 29.15A: Child with extensive superficial dermal burns with wet collagen dressing

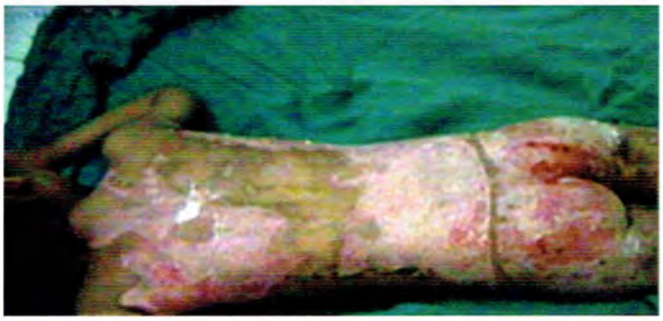

Fig. 29.15B: Same patient with healed areas in 10 days

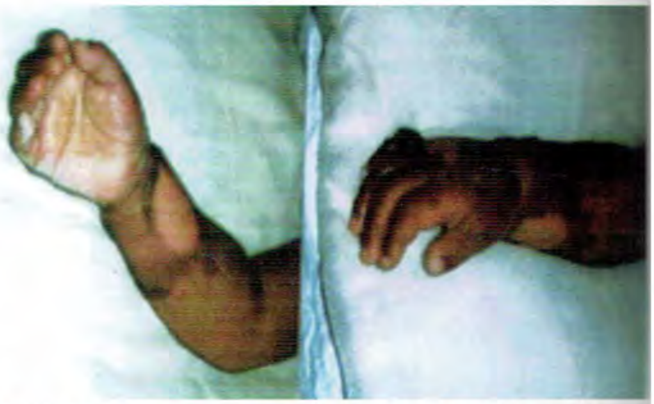

Fig. 29.16A: Superficial dermal burns of hand in forearm in an infa

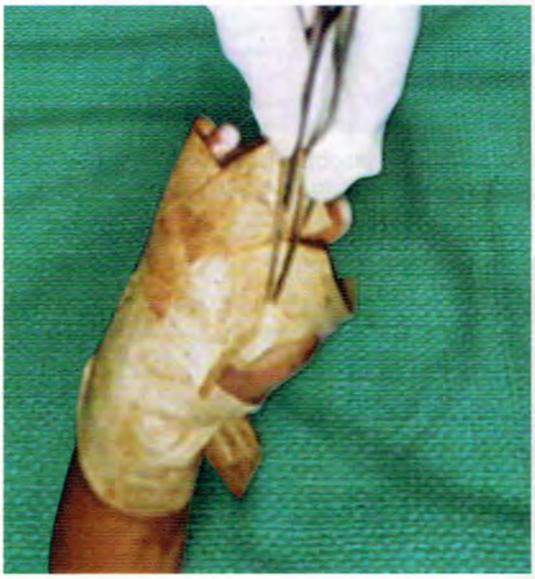

Fig. 29.16B: Same patient dressed with dry collagen (Skin Temp®)

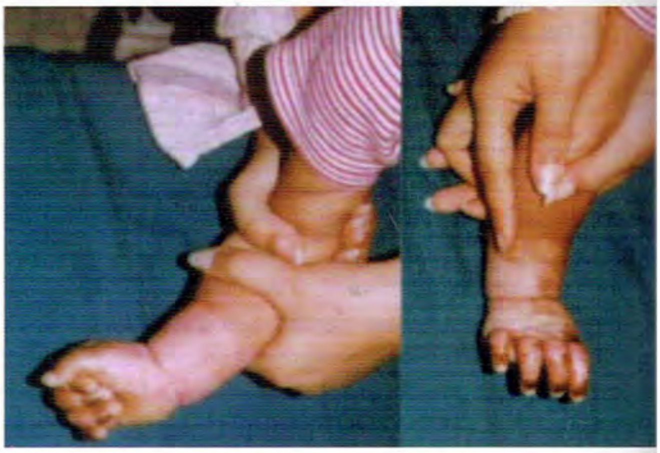

Fig. 29.16C: Healed area after 10 days

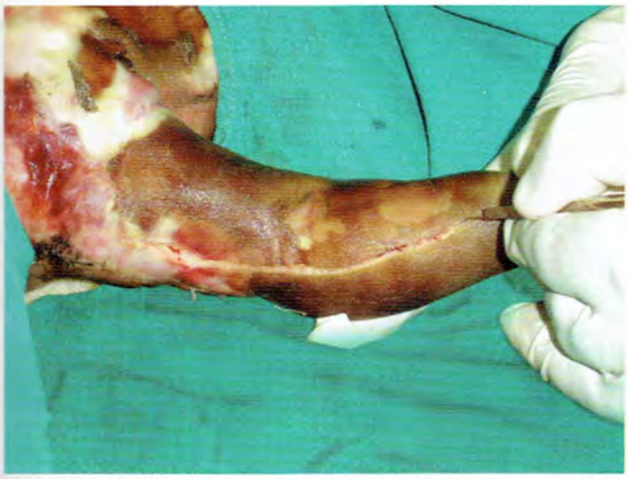

Fig. 29.17A: Escharotomy incision in full thickness circumferential burn in whole of the arm of an infant compromising distal circulation

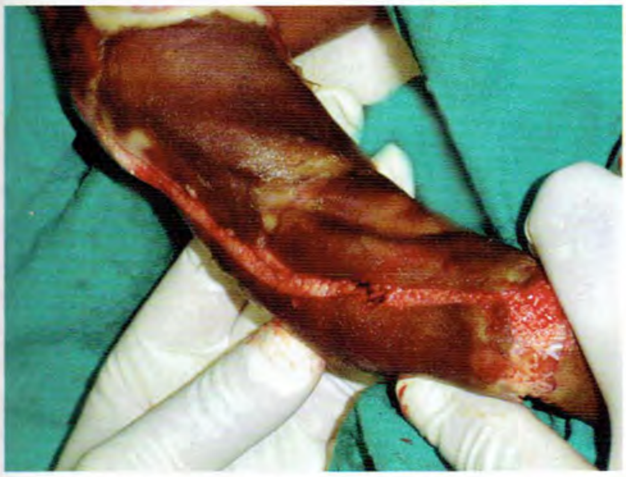

Fig. 29.17B: Circulation improved following lateral escharotomy incision

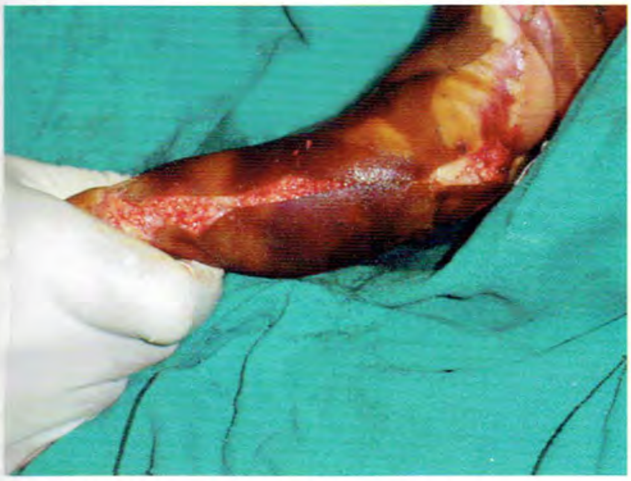

Fig. 29.17C: Medial escharotomy incision in same patient

In case of extensive deep burns in which patient cannot be taken up for early excision and grafting, the patient is treated by conservative management in the form of regular dressings till the eschar separates and the wound starts granulating (Figs 29.18A to 29.19C). In case there is deficiency of donor areas because of extensive wounds or in chronic neglected cases where the patient is cachexic and not a favorable candidate for auto-grafting, the raw areas can be covered with homograft till such time that the area can be covered by autograft in stages. Homograft taken from mother is preferred over other family members (Figs 29.20A to C). The best way is to apply alternate sheets of

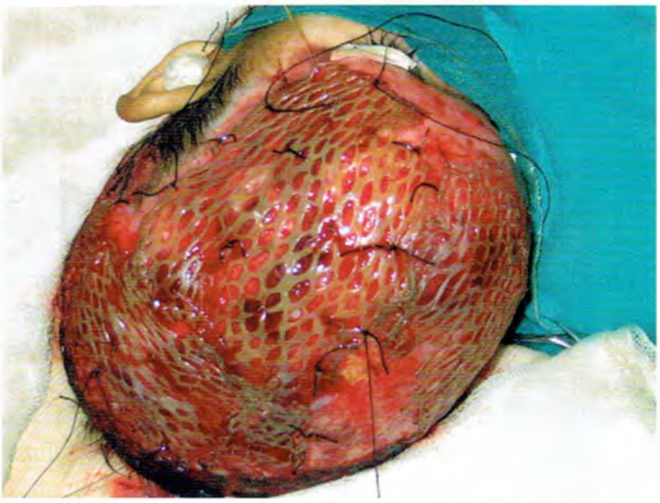

Fig. 29.18A: Split skin graft applied over post burn raw area over scalp in a 5-year-old child

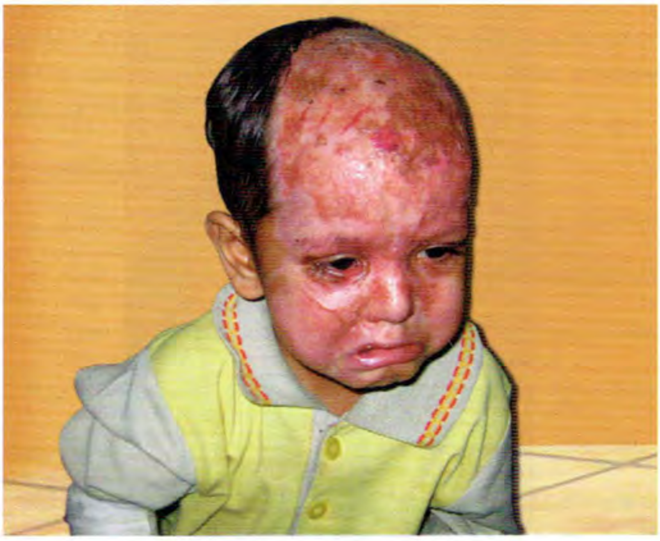

Fig. 29.18B: Completely healed scalp

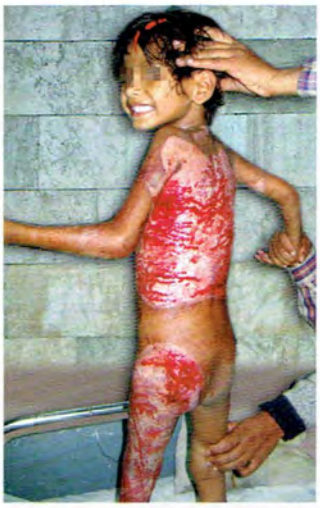

Fig. 29.19A: A 7-year-old girl with extensive post burn raw area after separation of eschar

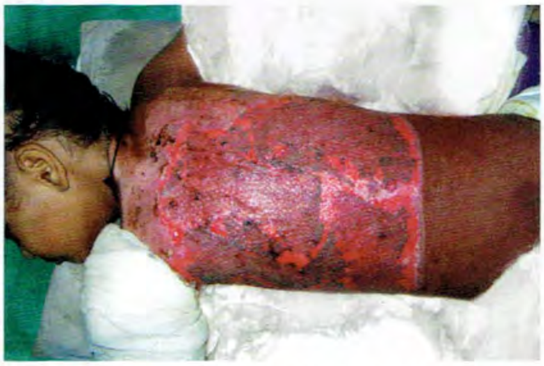

Fig. 29.19B: Same patient's wounds grafted as soon as granulation tissue was healthy

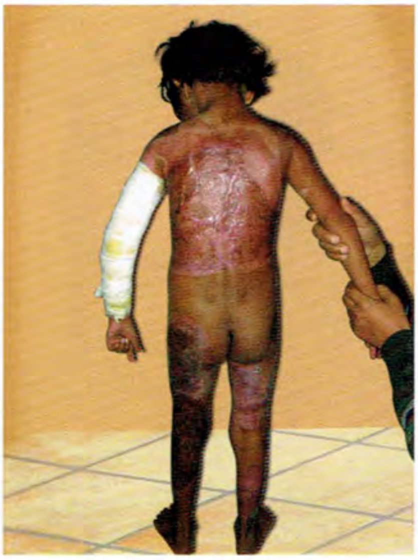

Fig. 29.19C: Healed areas after one month

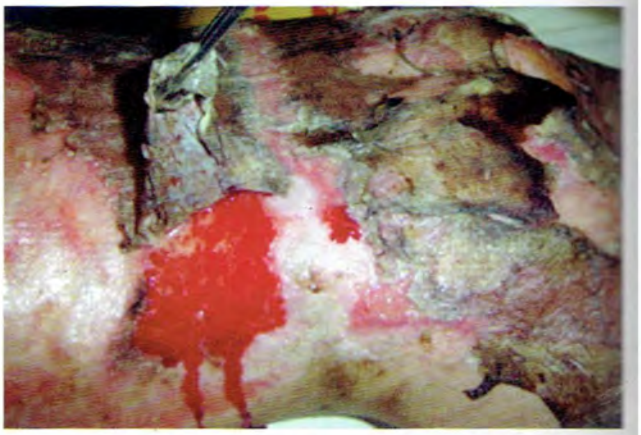

Fig. 29.20A: Homograft being removed after 7 days of application in a 2-year-old child with 50% burns with 25% raw area (mother of the child was the donor)

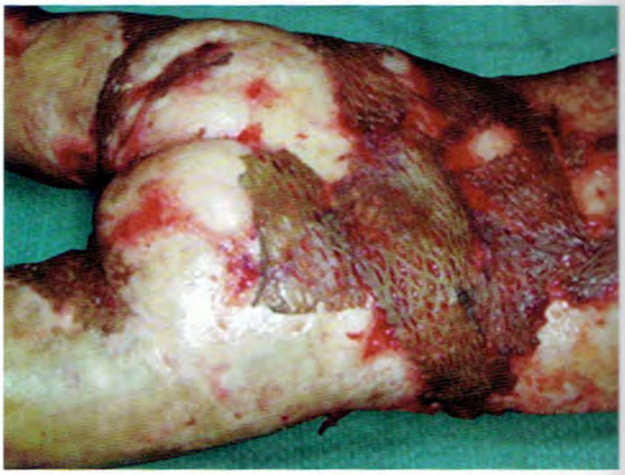

Fig. 29.20B: Autograft applied after removing homograft

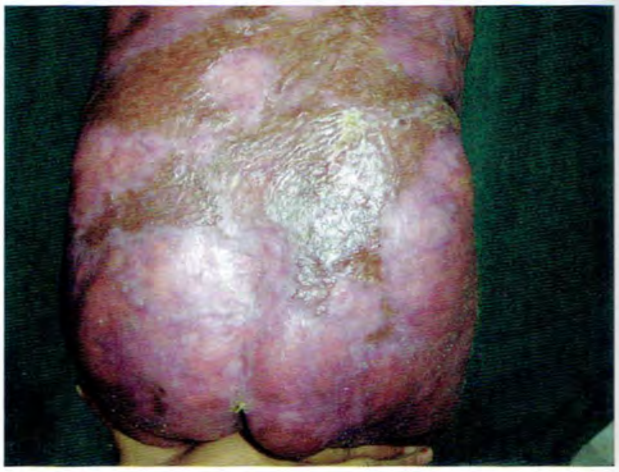

Fig. 29.20C: Same child with completely healed areas after 2 months

autograft and homograft. In such cases it is found that as the autograft starts spreading by epithelialization from the margins, the homograft starts separating automatically. But more commonly, it becomes necessary to excise the homograft before 2 weeks and replace them with autografts. This intervening 2-3 week period allows exceptionally good anabolic recovery of the child, improving not only the condition of the wounds but also the general condition of the patient.

Because of the paucity of epidermal appendages, the healing by epithelialization in case of deep dermal burns is much less in young children as compared to the older children and adults and usually they heal by migration of epithelium from the edges and contracture formation. In children less than 1 year of age, the usual tendency is to keep the limbs in an attitude of flexion so that at times the wounds heal by formation of flexion contractures. All these contractures should be prevented.

COMPLICATIONS

The most common complications of burns are burn wound infections and pneumonia which are leading causes of death in burnt children. Gastrointestinal complications are also common and include gastric ulceration (stress ulcer–Curling ulcer), pancreatitis, enterocolitis and superior mesenteric artery syndrome. Adequate fluid resuscitation is most effective means of prevention. Large surface area burns may cause hemolysis and muscle necrosis and lead to hemoglobinuria and myoglobinuria. Most often ignored are psychological sequelae because of disfigurement and loss of function.

PROGNOSIS

A large number of factors affect the prognosis in a burned child. Mortality in pediatric burns is affected by three main risk factors: inhalational injury, burn size more than 30% and age less than 4 years.[15] The presence of inhalational injury doubles the mortality rate. Management of extensive burns is extremely expensive and often prolonged. The poor socio-economic status is certainly a contributing factor in increasing the morbidity and mortality.

PREVENTION

Majority of burns in children can be prevented if adults become more careful and do not leave the children unattended. We should teach children about being careful of hot liquids, fire and electricity from a very young age (perhaps 2 years onwards). Since immersion scalds are very common while giving a bath to the child especially in winter, it is important for parents to ensure not to leave hot water bucket unattended and mix hot water into cold water while preparing for a child's bath and not vice versa. Firecrackers should be burst only under supervision of adults. The electrical sockets should be at a higher level and out of reach of small children. Strong acids and alkalis should be stored in houses at a place not accessible to children. Schools, television and other media can help in spreading awareness in the community. In India, there are no laws and regulations and strict enforcement of laws to prevent burns in childhood resulting in a very high incidence of burns. In developed countries like UK, there are a number of Acts, like 'The Children and Young Persons Act,1914', 'Heating Appliances Act,1971', Night Dresses for Children's Act,1962. etc. to safeguard against burns in children.

SUMMARY

Burns in children leave lifelong scars which are enormous stressors for both the child and his parents as they affect their development not only physically but mentally as well. Burn management in pediatric age group can be a great challenge because physiologically, the skin and organ systems are immature and cannot tolerate stress like an adult and therefore, they respond differently to thermal injury. Emotionally, children are again immature and incapable of handling pain and disfigurement associated with burn. The burn wound management and the surgical approach to pediatric patients, therefore, varies according to depth and extent of burns. Both the child's family and the treating doctor have an important role to play in the eventual outcome of the child who comes out successfully from the physical trauma of burns.

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Chapter 30

Burns during Pregnancy

Shailesh Ranade

INTRODUCTION

Burns during pregnancy presents a unique medical problem. Both mother and fetus are at great risk of fluid loss, hypoxemia and sepsis. Most of the textbooks on obstetrics do not deal with burns in pregnancy nor is the topic considered in depth in books devoted to the treatment of burns. In India and other countries, where burns present as a socio-medical problem, the incidence of burns in pregnancy is far more than that in the Western world.

INCIDENCE

The literature on burns during pregnancy is quite limited. However, the incidence calculated by the various authors in relation to the total number of female burn patients of reproductive age, does not appear to be low. The incidence in early pregnancy was unknown in past due to the absence of routine pregnancy testing on admission. Easy availability of pregnancy determination tests has increased the reporting of burns in pregnancy.

The highest incidence of burns during pregnancy has been reported from India. It has ranged from 7% as calculated by Akhtar [1] to 13.3% reported by Jain [2] and 15% reported by Prasanna.[3] Taylor[4] calculated an incidence of 7% while Amy[5] (1985) reported the incidence as 6.7%. Similar incidences have been reported by Gang [6] from Kuwait (7.8%) and Srivastava [7] (7.9%). Benmeir [8] from Israel reported an incidence of 0.6%.

ETIOLOGY

The aetiology of burns in pregnancy does not vary from the general etiology of burns as a whole. A special mention is, however, required for burns in pregnancy in countries like India, where dowry and familial disputes in a joint family are common causes of suicidal and homicidal burns. These are wrongly reported as accidental burns due to false history given by the victim. Most of these patients are very severely burned and often have a very high maternal and fetal mortality.

PATHOPHYSIOLOGY OF BURNS IN PREGNANCY (FLOW CHART 30.1)

A pregnant burned patient presents following additional pathophysiological changes.[9,10]

1. Pregnant patients have a lower FRC (Functional Residual Capacity) and are less able to compensate for hypoxia. Moreover, exposure to burns often puts the mother at risk for carbon monoxide inhalation. The fetus is especially susceptible to carbon monoxide poisoning because fetal hemoglobin has a higher affinity for carbon monoxide than maternal hemoglobin does. Thus, carbon monoxide can easily lead to fetal hypoxia.
2. *Effect of Burns on Uterine Activity:* The onset of labor in a premature delivery shortly after a serious burn was in the past thought to be triggered by the endocrine function, especially by secretion of adrenocortical hormones related to stress. Later observations confirmed that minor burns had no effect on the course of pregnancy, while bums of at least 35% TBSA were capable of provoking early labor and the loss of the fetus following intrauterine death within a week of the burns.

Both spontaneous miscarriage and premature delivery were subsequently thought to be related to the synthesis and release of prostaglandins (responsible for early uterine contractions) from the skin in the burned area. Later, however, the correct importance was attributed both to maternal shock, which determines a considerable reduction in the uterine blood flow and causes fetal hypoxia and to pleuropulmonary complications, especially in cases of inhalation injuries with grave reduction of maternal pO_2 and consequently fetal hypoxia. The fetus may be able to tolerate the early phases of maternal sepsis but is notably affected in the advanced phases, when the mother is decompensated and her cardiovascular system collapses. Thus, hypotension and acute respiratory insufficiency with septicemia can lead to complications in the fetus, even some time after the burn (Flow chart 30.1)

As the maternal intravascular space is in a state of equilibrium with the amniotic liquid, the reduction of this liquid during serious hypovolemia can cause the intrauterine death of the fetus. The onset of spontaneous uterine contractions is also favored by the release from bacteria and the placenta of an enzyme, phospholipase A, which is necessary for the conversion of arachidonic acid into prostaglandin. It has recently been shown that there is a considerable reduction in plasma levels of 17β-estradiol in pregnant burned women who had either an abortion or a still birth in the first week post burn (Figs 30.1 and 30.2).

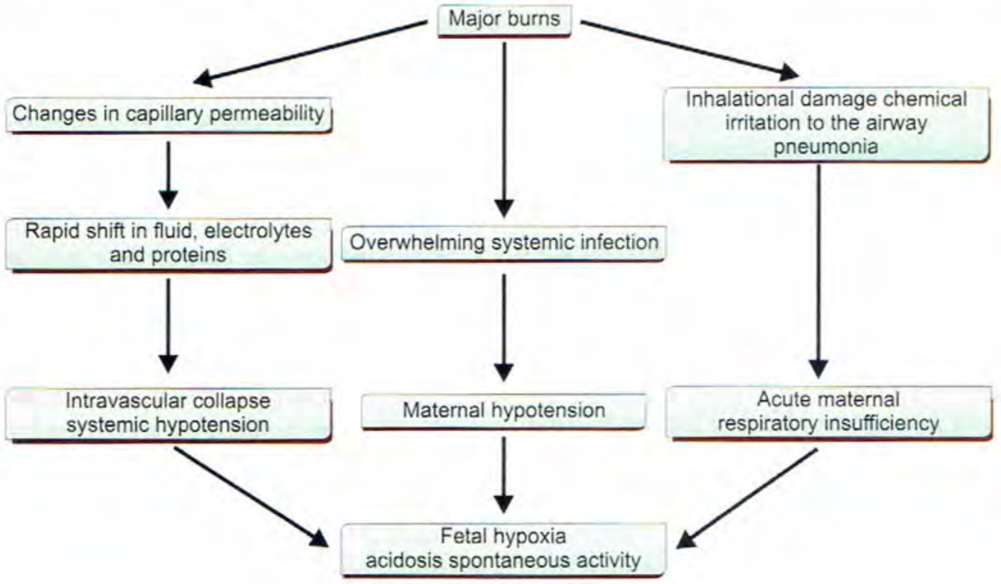
MANAGEMENT OF BURNS AND ITS INFLUENCE ON THE COURSE OF PREGNANCY AND THE FETUS

Medical Treatment

Resuscitation treatment in the burned pregnant woman is no different from that in the non-pregnant burned woman. The prevention of hypovolemic shock by adequate early fluid resuscitation, therefore, requires that the uterine blood flow should be able to maintain fetus tissue pO_2 levels within the normal range. It has been recommended that a quantity of fluid should be administered that is sufficient to maintain the mother's blood pressure within the normal range and maintain urine output to 0.5-1.0 ml/kg body weight/hour. The maintenance of arterial pressure levels at normal values is essential at all stages of the burn disease. Diuretics and antihypertensive drugs should, therefore, be avoided, whenever possible. Episodes of hypotension should also be avoided in the event of surgical operations. It is recommended that surgery should be performed with intraoperative maintenance of a minimum of 1 ml/kg body weight/hour of urine volume and 100% oxygen saturation.

Since extensive surface burns are frequently associated with an increased rate of arterial shunting and hypoxia, it becomes necessary to administer oxygen. A pregnant patient's oxygenation can often be improved by nursing her in a semi-sitting position. Maternal pO_2 values of less than 60 mm of Hg during the pleuropulmonary complications

Flow chart 30.1: Pathophysiological changes in pregnant patient following major burns, affecting the foetus (*Courtesy:* Rayburn, 1984)


Major burns
Changes in capillary permeability
Inhalational damage chemical irritation to the airway pneumonia
Rapid shift in fluid, electrolytes and proteins
Overwhelming systemic infection
Intravascular collapse systemic hypotension
Maternal hypotension
Acute maternal respiratory insufficiency
Fetal hypoxia acidosis spontaneous activity

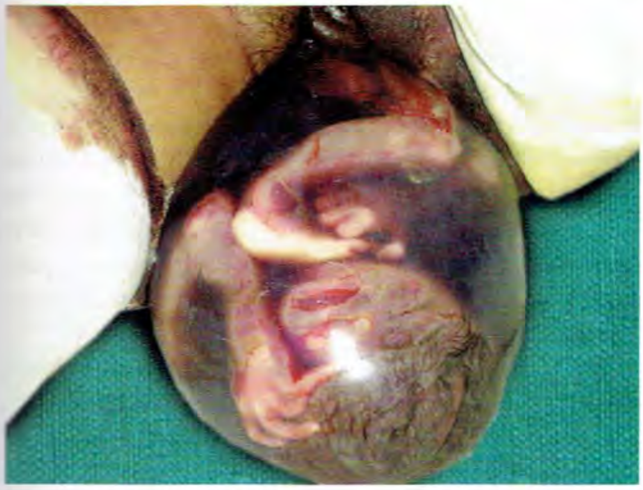


Fig. 30.1: Patient with 90% thermal burn and 7 months pregnancy aborted on the third day post burn spontaneously. Aborted fetus seen within intact membranes

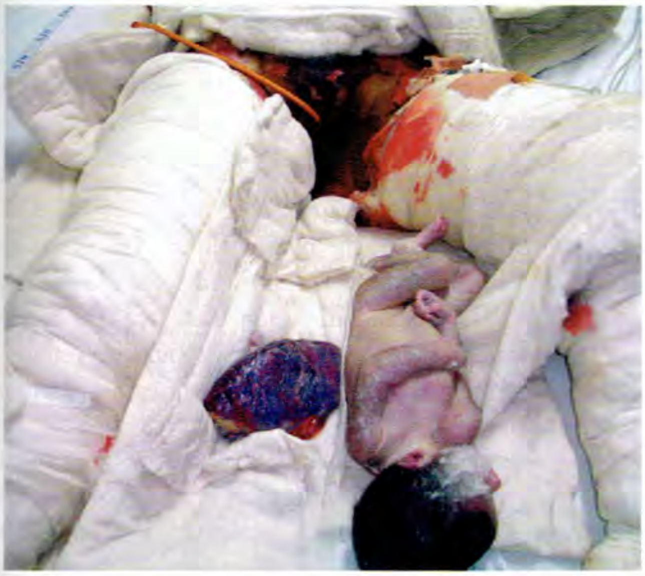


Fig. 30.2: Patient with 60% thermal burns and full term pregnancy was induced within 48 hours to deliver a normal live baby as seen in the picture

that are often secondary to inhalation have been considered critical. It has, therefore, been recommended that ventilatory support should be initiated as soon as possible. This is all the more necessary because inhaled carbon monoxide can also cross the placental barrier to compete for binding sites on fetal hemoglobin, provoking fetal cardiac edema and also affect cardiac development. The newer inventions in respiratory supportive care, like BIPAP (Biphasic Positive Airway Pressure) and CPAP (Continuous Positive Airway Pressure) ventilators have added a non-invasive method of ventilation as an option to standard ventilators. In cases of bronchopneumonia, it is necessary to use antibiotic treatment, if possible, by selecting drugs that the fetus can tolerate.

Wound Care

The local treatment of burns in pregnant women is not simple, because of the limitations imposed by the state of pregnancy.

- Sulfa drugs (silver sulfadiazine) are suspected of potential teratogenous activity if administered before the 14th week of pregnancy and of retarding growth, determining low birth weight if subsequently administered. Sulfa drugs administered at the term of pregnancy are responsible for kernicterus.
- Povidone-iodine is widely used for burns cleansing. It must, however, be avoided during pregnancy since large amounts of iodine can be absorbed through the burn wound. Use of povidone-iodine is inadvisable in burns exceeding 20% total body surface area (TBSA) because the iodine passing through the placenta can be absorbed in sufficient quantities to affect thyroid functions and cause metabolic acidosis.

  It is, thus, clear that the state of pregnancy considerably reduces use of the commonest protocols for the topical treatment of burns.
- Salicylates exert an anti-prostaglandin action and may, therefore, have a protective effect on pregnancy. For the same reason they should not be used in the final period of pregnancy because they prolong gestation and delay spontaneous delivery. There is little evidence regarding the possibility that salicylates have a teratogenous effect, although the birth weight of babies born to women subjected to chronic administration of salicylates has been reported to be below average. An increase in perinatal mortality has also been reported. These results have not, however, been confirmed in other studies.
- Chloramphenicol, either in powder form or as an ointment, is among the drugs to be avoided throughout pregnancy since it is teratogenic if administered during the first period of pregnancy and responsible for neonatal pathology if used during the final period (Grey syndrome).
- Gentamicin (ointment form) is an aminoglycoside capable of passing through the placenta after absorption

through burned area. If used after the 14th week of pregnancy, it can cause lesions in the 8th pair of cranial nerves, with vestibular and acoustic damage. The full-term fetus has been found to present antibiotic concentrations analogous to those of the mother. In addition aminoglycosides are responsible for nephrotoxicity.

- Rifamycin (for local application) is absolutely avoided during the last period of pregnancy because its interference with bilirubin metabolism can cause indirect hyperbilirubinemia with risk of kernicterus.
- Even the local use of antibiotics that are normally administered systemically presents considerable difficulties.

The only antibiotics that can be considered safe in pregnancy are the penicillins and cephalosporins, while vancomycin, one of the most frequently used and most active antistaphylococcal drugs, is considered to be potentially teratogenic and ciprofloxacin may possibly damage articular cartilages in undeveloped organisms.

Other commonly used antibiotics (Imipenem, teicoplanin) should be avoided in the absence of adequate information about their safety during pregnancy unless they are absolutely necessary and their advantages outweigh any possible risk.

Tetracyclines are not widely used in burns but it should not be forgotten that they are contra-indicated during pregnancy because of their varying effects on fetal growth, bones, teeth and the immune system.

With regard to surgical procedures performed under anesthesia, it is important to avoid the use of ketamine, which increases the excitability of the myometrium because it is capable of triggering effective contractions. When the pregnancy is near term and delivery is imminent, ketamine may cause respiratory depression in the neonate.

It is preferable to keep the operative procedures as short as possible. During the period of anesthesia, it is important to maintain the oxygen saturation and blood pressure within normal limits throughout the procedure.

Surgical Treatment

In view of the difficulties related to local medical treatment, early surgical treatment assumes vital importance. This is because early surgery facilitates healing of the wounds and thus improves prognosis of both mother and neonate. Early burn wound cover also minimizes septic complications and the need to administer antibiotics; in addition, it reduces the necessity of analgesic drugs.

The treatment for full thickness burns comprises early tangential excision and split-thickness skin-grafting 3-7 days post burn (but not more than 15-20% TBSA at one operation). The invention of biological skin substitutes has dramatically improved the management of II° burns. Processed animal collagen sheets (Commercially available as Apocoll and Kollagen), Integra, Biobrane help in covering even very large burn wounds preventing the fluid loss, reducing pain, chances of wound infection and obviating the need of regular dressings.

Wounds over the abdomen and breast have to be treated first. Good early healing of the abdominal wound favors:

- Pain-free stretching of the abdominal skin during the developing pregnancy to term
- Abdominal obstetric supervision of the growing fetus
- Performance of cesarean section if required

Early surgery of the breast wound prevents infection and sloughing of nipples and permits subsequent breastfeeding.

Obstetric Management of the Pregnant Woman with Burns (Table 30.1)

If it is known that a burned female is pregnant, it is important to establish as precisely as possible the exact stage of pregnancy at the time of the burn accident. This must be based upon the menstrual history and fetal ultrasound examination. The gestational period is in fact one of the factors determining obstetric procedures (no intervention, protection of pregnancy by tocolytic treatment, induction and/or acceleration of labor). Other factors such as the severity of the burn and fetal viability must be confirmed immediately. Ultrasound examination helps in anatomical measurements such as biparietal diameter, to determine the age of fetus, biophysical measurements such as fetal muscle tone, limb motion and breathing patterns to know the condition of fetus. In addition placental morphology, placental blood flow and amniotic fluid volume may be visualized in order to assess fetal health.

With regard to the stage of gestation, fetuses delivered before 24 weeks generally do not survive, while those delivered after 32 weeks do well with modern neonatal intensive care if born without hypoxia or birth trauma. The most difficult to manage are fetuses between 24 and 32 weeks' of gestational age, where *ex utero* survival is difficult to predict. In such cases, therefore, when preterm labor occurs, tocolytic procedures are initiated.

Table 30.1: Obstetric management in burned patients[6]

Total % burn	*Age of Gestation*	*Management*
< 30 % TBSA	First trimester	No obstetric interference
	Second trimester	No obstetric interference
	Third trimester, less than 36 weeks	Conservative approach and monitoring of heart rate
	Third trimester, more than 36 weeks	Induce labor/Cesarian section
30-50% TBSA	First trimester	Fetal monitoring by ultrasound 3-4 weeks
	Second trimester	Fetal monitoring every 3-4 weeks. Tocolytic therapy
	Third trimester, less than 36 weeks	Careful fetal monitoring
	Third trimester, more than 36 weeks	Deliver fetus within 48 hours
50-70% TBSA	First trimester	Terminate pregnancy
	Secotd trimester	Terminate pregnancy
	Third trimester, if baby is viable	Induce labor/cesarian section within 24 hours.
	Third trimester, intrauterine death	No active intervention up to 4 weeks/monitoring of hemocoagulation factors
> 70% TBSA	First trimester	No treatment
	Second trimester	No treatment
	Third trimester	Cesarian section as an emergency procedure at the earliest

In women with advanced state of pregnancy (2nd and 3rd trimester) with more than 50% TBSA burns, it had an unfavorable effect unless delivery was immediate, as the burn created an unfavurable environment for the fetus. In burns less than 40% TBSA, pregnancy and its continuation had no effect on prognosis in the mother and every attempt had to be made to interrupt inception of labor if the fetus is too immature to survive.

Regarding the manner of delivery (vaginal route, cesarean section), spontaneous vaginal delivery is generally preferred, although obstetric considerations affect the choice of route and the timing of the delivery. Serial fetal sonography and fetal heart rate monitoring identifies fetal stress at an early stage and may permit timely intervention, preventing intrauterine death. In a critically burned woman with a living and near-term pregnancy, fetal salvage by cesarian section appears justifiable.

When there are obstetric indications for a cesarean section, this can be performed even when the lower abdominal wall is part of the burned area.

Thus, pregnant burned patient, especially when severely burned, is a big challenge for a Burn Surgeon. A comprehensive team effort involving a Plastic Surgeon, Intensivist, Obstetrician, Neonatologist, Anesthesiologist, Chest Physician and well trained nursing staff with other supportive departments can help in saving two lives at a time.

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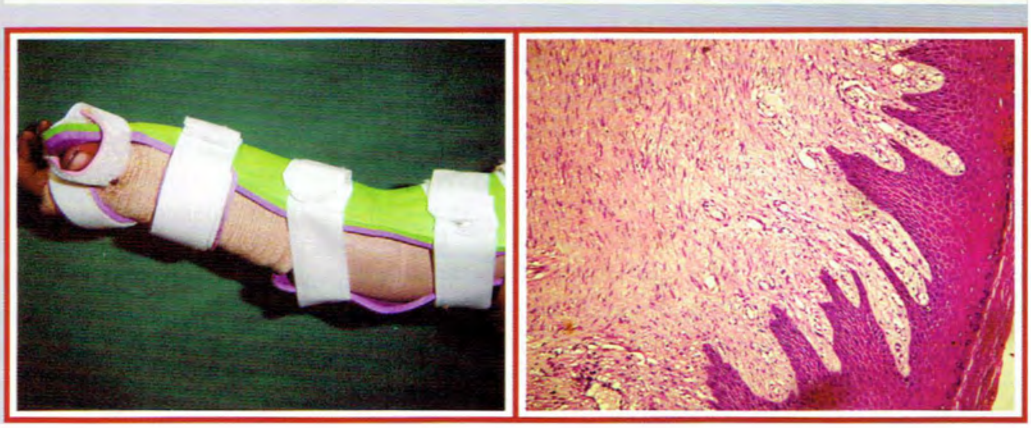
6 Section

General Management and Rehabilitation



Chapter 31

Multiple Organ Dysfunction Syndrome in Burns

Anil Chadha, Indu Chadha, Amitabh Singh

INTRODUCTION

"The body falling part, mind failing, spirits weakened and the will lacking – all heralding grave tiding" conveys the sense of hopelessness Multiple Organ Dysfunction Syndrome reflects.[1] MODS or Multiple Organ Dysfunction Syndrome is the major cause of mortality in extensive burns.[2,3] It has been known for more than three decades but an assured defense against it is yet to be convincingly established. As has been understood since long, the management of major burns is mainly an exercise in combating the systemic inflammation precipitated by the burn trauma. The inflammatory reaction of the body is the physiological basis for recovery from such a trauma. It is the self perpetuated excessive host defense that proves counter-productive in some patients and leads to a state, wherein there is dysfunction and eventual failure of one or more organs, called MODS. The ability to prevent/treat MODS is the defining point in the management of critical burns. This chapter attempts to explain the meaning of MODS and its significance in context to burns. The essence of the reactions to the biochemical mediators of inflammation has been mentioned without mentioning the details of each one in the cascade in order to relate to this complex process with burns in an undiluted manner.

As a prelude to the recognized entity of MODS, Tilney et al (1973) reported the failure of multiple organs in the postoperative period of ruptured aortic aneurysm.[4] Interestingly, a similar clinical picture was seen in diverse conditions like pancreatitis, peritonitis, bowel perforation, prolonged shock following valve replacement and major burns by Baue in 1975.[5] It was termed *'Sequential System Failure'*. In 1977, Eiseman, Beart and Norton named it *'Multiple Organ Failure'*.[6] Fry and his colleagues in 1980 noticed that gram-negative sepsis was a common event that preceded MOF due to the diverse causes.[7] The lipopolysaccharide (LPS) was identified as the responsible bacterial endotoxin and from 1980 to 1985, empirical treatment of infection with antibiotics was the mainstay of therapy in multiple organ failure.[7, 8] It was later realized that not all patients of MODS were due to infection and those dying of MODS did not always have culture proven infection.[7] Search for a common mechanism to the diverse etiologies of MODS is still elusive and the molecular basis for specific organ failure remains to be fully uncovered.

The clinical entity of MODS has come into being with the prolonged survival of the critically ill patients due to the advances in critical care in the last three decades.[9] Earlier these patients with multiple failing organs would surely have died. Research in this field has improved the understanding about the course of events and it has prompted promising therapeutic interventions. Before understanding the pathological basis of MODS, a brief overview of the course of events in MODS would help us understand the condition in a better context.

Since 1800s it has been known that a burn injury is an inflammatory reaction.[10] It is typically a defense reaction for the benefit of the body. It remains localized as long as the area of burns is small and the inflammatory state abates with the healing of the wound. In instances where the area of burns is large (> 20% TBSA), the body responds with an inflammatory reaction of a larger magnitude. This state is mainly homeostatic and immunological in the immediate setting with other processes following for the defense of the body. A state of systemic inflammation sets in as a local defense and homeostatic control (Fig. 31.1). Adequate resuscitation at this stage potentially reverses the deranged systemic changes due to the burn trauma. However, due to large extent of burn trauma, the hypermetabolic state has been seen to persist even in the absence of any complications or infection and sometimes even after the burn wound has healed.[11] The biochemical mediators that are released due to the inflammatory/immune response and the neuroendocrine activity, act for the benefit of the host defense and repair mechanism. The presence of necrotic slough, uncovered wounds, donor site raw areas, inadequate resuscitation, pathological fluid derangements, hypoperfusion of gut mucosa, nosocomial infections, access port infections, stress response, reperfusion injury, etc. perpetuates this state of systemic inflammation. These small but persistent stimuli along with the high risk of wound contamination to invasive infection link up to generate a momentum in the systemic inflammatory state, which gets detached from the control of counter-inflammatory measures and becomes uncontrolled and self perpetuating. The local and homeostatic responses become systemic derangements exacerbated not only by endothelial damage but also ischemia/reperfusion injury.[12]

As a protective reaction, the inflammatory process induces vasodilatation and increased capillary permeability at the local site of burn injury. When these mediators come into systemic circulation it results in systemic vasodilatation and leakage of intravascular fluid into the third space resulting in pulmonary edema, generalized edema and severe hypotension. This nonspecific activation of a generalized inflammatory response mainly from leukocytes, macrophages and endothelial cells results in a sequence of events that leads to intravascular microthrombi and the inability to use the available oxygen, which marks the progression of a patient from SIRS(Systemic Inflammatory Response Syndrome) to MODS. MODS encompasses failure of one or more organs, most commonly the lungs; it deteriorates from its optimal function and may progress to failure. Since it is a dynamic change rather than an 'all or none' phenomenon, the term MSOF (Multi System Organ Failure) has been replaced by MODS Multiple Organ Dysfunction Syndrome).[13]

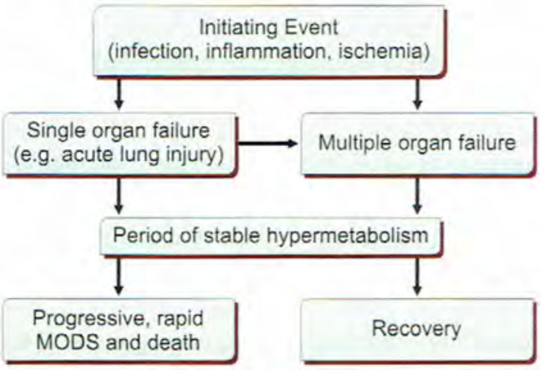


Fig. 31.1: Natural course of MODS

The already decreased circulation due to the fluid shift is further compromised by the formation of microthrombi; resulting in global tissue hypoxia. Hypoxia activates the sympathetic nervous system causing the stress response,[14] which in essence shunts more blood to the heart depriving other organs of oxygen, mainly the kidneys and gut.

Hypoperfusion of the gut, besides facilitating bacterial translocation, results in release of pro-inflammatory mediators, which contributes to the mediator-induced acute lung injury, bone marrow depression, myocardial dysfunction, neutrophil activation, red blood cell injury and endothelial cell activation and injury.[15] Compromised renal function, due to hypoperfusion, impairs its ability to remove toxins from the body and makes their function as an acid-base buffer system ineffective at a time when it is more efficiently required. Generalized hypoxia leads to anaerobic metabolism and consequent lactic acidosis. The increased lactate levels further exacerbate MODS by altering the intracellular ion pumps, mitochondrial functions and normal carbohydrate metabolism. The adrenergic activity of the stress response leads to retention of sodium and water, hyperglycemia and insulin-resistance. The function of the other acid-base buffer system of the body, the lungs, is already compromised due to the mediator-induced injury as the lungs are more susceptible. Interestingly, the usual sequence of organ dysfunction in progression from SIRS to MODS is lungs, gut, central nervous system, vasomotor, cardiac, hematologic, liver and then renal.[16,17] To further compromise the functioning of the body, the fluid shifts to the third space result in pulmonary edema and further decreases oxygenation at a time when it is most required. At this juncture the autonomic dysfunction due to MODS disjoints the cardio-respiratory system.[18]

All these events occur almost in tandem with the clinical progress of the burn wound and the burnt patient. As long as the majority of the areas are timely excised and covered after efficient 'goal directed' resuscitation in a patient, who is low risk for progressing to MODS; the wound heals and the patient recovers. On the contrary, a similar patient with all the risk factors would probably end up with a prolonged inflammatory state with high likelihood of progressing to MODS (Fig. 31.2) if due attention to its prevention is not given. It is imperative to understand in treatment of organ support and MODS that the unregulated host response rather than the triggering factor has the main bearing on the outcome. Further sections detail the pathogenesis along with the predisposing risk factors and the methods to monitor the patient so that any deterioration or progression towards MODS could be detected at the earliest and corrective intervention prompted. Hence, the importance of a dynamic scoring system for MODS in burn trauma with high sensitivity and specificity to diagnose the severity/ reversibility of organ dysfunction, day-to-day monitoring and if possible, predict mortality.

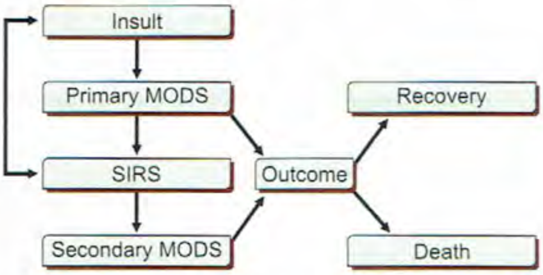


Fig. 31.2: Outcome of MODS

Unfortunately, the quantum of research in MODS is mainly related to the non-burn population and hence a consolidated protocol for each event is still in evolution. The most promising therapy for MODS, even today, is its prevention.[19] MODS has a reported mortality of more than 50%[20,21] which increases to 60 to 98% with the number of organs failing and the duration they remain dysfunctional.[22] Treatment protocols include organ support, alleviation of the trigger factors and an attempt to counter the unwanted effects of the mediators of inflammation, which have yet to significantly improve the outcome in critical burns.

COMMON CHEMICAL MEDIATORS

A very brief outline of the common chemical mediators is mentioned here for ease of understanding; it is not a complete description.

Endotoxins:[23,24] This is the lipopolysaccharide component of the outer membrane of the gram-negative bacteria. It stimulates the release of the biochemical mediators of inflammation and triggers the complement cascade. When this beneficial inflammatory response to the endotoxin becomes unregulated, it may lead to endothelial damage of capillaries resulting in interstitial edema and organ dysfunction.

CYTOKINES:[25] Briefly cytokines have the following functions (1) directing a T lymphocyte response; (2) inducing enzyme production in distant sites (e.g. endothelium: nitric oxide, liver: C reactive protein); (3) altering cell surface adhesion molecules.

They are categorized as (a) growth factors (e.g. Transforming growth factor β); (b) leukocyte chemotactic chemokines (e.g. IL-8); (c) modulators of lymphocyte function (e.g. IL-4) and (d) modulators of inflammatory response (IL-6 as a pro-inflammatory mediator, IL-10 as an anti-inflammatory mediator). Cytokines induce MODS by (1) excessive production of free radicals (2) induction of elastase or endopeptidases and (3) elevation of circulating soluble peptides that activate apoptosis (programmed cell death). TNF-α and IL-1 are considered the most essential mediators responsible for MODS.[26] They activate the immunological activity and endothelial cells, inducing fever and excessive hypermetabolism besides causing local tissue destruction, microvascular injury and increase in microvascular permeability leading to hemodynamic insufficiency and a refractory shock state.[27]

TNFα[28]—An amino acid polypeptide released as an inflammatory reaction from monocytes, pulmonary macrophages, Kupffer cells and endothelial cells; it has a pleiotropopic effect on the target cells. Notable deleterious effects are induction of fever, SIRS, myocardial depression and cardiovascular collapse, shock, elevated liver enzymes, coagulopathy and carbohydrate, fat and protein metabolism leading to cachexia.

Interleukin-1[29]—They are expressed from monocytes, tissue macrophages, neutrophils and B lymphocytes during the inflammatory response. IL-1, present as IL-α and IL-β, accelerates the T cell proliferation and induces mature and immature granulocytes from the bone marrow. It also directly stimulates neutrophils and is a strong inductor of granulocyte/macrocyte colony stimulating factor (CSF) and CSF-release. It causes fever, hemodynamic abnormalities, anorexia, arthralgia and headache by stimulating local release of prostaglandins from the anterior hypothalamus.

Interleukin-6 – IL-6 is a glycoprotein, produced by lymphocytes, fibroblasts and monocytes. It activates B and T lymphocytes, protein production in liver and coagulopathy. IL-6 is an integral part of cytokine cascade in organ injury.

Interleukin-8 – It is protein cytokine produced by mononuclear phagocytes, PMN leukocytes, endothelial cells, epithelial cells and mesothelial cells in response to endotoxin, IL-1 and TNF-α. IL-8 attracts and activates neutrophils to the site of inflammation and is an angiogenic factor. It causes transient neutropenia but no tissue neutrophilia, inflammation or injury has been seen with IL-8.

Soluble receptor and receptor antagonists – sTNFR - Soluble cytokine receptors produced by proteolytic cleavage of cell surface have been reported for TNF-α, IL-6 and IL-1β. sTNFR has ability to bind TNF-α and mediate the actions of the cytokines. The bioactivity of TNF-α is based on the equilibrium between free TNF-α and that bound to sTNFR. The IL-1 receptor antagonist concentration is seen to increase in infection; however it does not correlate with the outcome.

Adhesion molecules—The diapedesis of neutrophils from the intravascular space to the site of inflammation is effected by loose tethering (Primary adhesion), which either may flow away with the blood stream or develop into strong binding (Secondary adhesion) to the endothelium; a prerequisite for migration into the extravascular space. These adhesion molecules are induced by cytokines like TNF-α and IL-1.

Nitric oxide (NO)[30]—TNF-α and endotoxins can induce calcium independent synthesis of NO. It causes vascular smooth muscle relaxation and inhibition of platelet induced thrombosis, myocardial depression, cytotoxic effects and organ failure. Activated macrophages and kupffer cells are said to produce NO in shock state.

Arachidonic acid metabolites – They are released from leukocytes, macrophages and pulmonary endothelial cells via the cyclo and lipo-oxygenase pathways. These metabolites can modify the host response in septic shock. Thromboxane induces potent vasoconstriction, bronchoconstriction and platelet aggregation. Leucotrienes are implicated in ARDS and they participate in pulmonary vasoconstriction, bronchoconstriction, allergy, coronary vasoconstriction and myocardial depression.

PAF – It is an active phospholipid synthesized normally in the body. It is produced by cytokines, macrophages, neutrophils, eosinophils, endothelial cells and platelets, kidney glomerular cells and gastrointestinal epithelial cells. PAF is a critical mediator in septic shock, ARDS, graft rejection and MODS by causing increase in vascularity, permeability and causes endothelial bed leak and platelet aggregation. Pro-inflammatory effects of PAF cause neutrophil priming and PMN endothelial adherence, which are responsible for the distant organ injury.

The following text describes the etiopathology of MODS in Burns, the scoring systems and the management. To help understand multiple organ dysfunction syndrome in context to burns it is imperative that one follows a standard definition to label an organ as dysfunctional or a patient to be in SIRS, etc. Hence, a brief summary of the definitions of organ failure, SIRS (Systemic Inflammatory Response Syndrome), MODS and sepsis is being mentioned.

ORGAN-SPECIFIC DEFINITIONS OF FAILURE[39]

If the patient had one or more of the following during a 24 hour period (regardless of the values), OSF existed on that day

Cardiovascular failure (presence of one or more of the following)
Heart rate ≤ 54/min
Mean arterial blood pressure ≤ 49 mm Hg
Occurrence of ventricular tachycardia and/or ventricular fibrillation
Serum pH ≤ 7.24 with a $PaCO_2$ of ≤ 49 mm Hg

Respiratory failure (presence of one or more of the following):
Respiratory rate ≤ 5/min or ≥ 40/min
$PaCO_2 \geq 50$ mm Hg
$AaDO_2 \geq 350$ mm Hg ($AaDO_2 = 713\ FIO_2 - PaCO_2 - PaO_2$)
Dependent on ventilator on the fourth day of OSF, e.g. not applicable for initial 72 hours of OSF

Renal failure (presence of one or more of the following):
Urine output ≤ 479 ml/24 hour or ≤ 159 ml/8 hour
Serum BUN ≥ 100 mg/dl
Serum creatinine ≥ 3.5 mg/dl

Hematologic failure (presence of one or more of the following):
WBC ≤ 1000 mm^3
Platelets ≤ 20,000/mm^3
Hematocrit ≤ 20%

Neurological failure
Glasgow Coma Score ≤ 6 (in absence of sedation at any one point in the day)

Sheridan[17] simplified the definitions of organ failure in his review, they are as follows:

Organ system	Definition
Lung	Ventilator dependence more than 72 hours after injury or PaO_2/FiO_2 ratio ≤ 150
Gut	Ileus requiring cessation of tube feedings
Central Nervous System	Glasgow Coma Score ≤ 6 in the absence of neuromuscular blockade or significant sedation
Vasomotor	Requirement for alpha vasopressors to maintain normal mean arterial pressure
Cardiac	The requirement for Inotropes or chronotropes or a serum pH ≤ 7.24 with serum $PaCO_2 \leq 49$
Hematologic	Platelet count ≤ 100,000 or WBC count ≤ 3500 per ml.
Liver	Serum transaminases ≥ 1.5 of normal maximum value or PT and PTT ≥ 1.5 control with bilirubin ≥ 5.0
Renal	Serum BUN ≥ 100 and creatinine ≥ 3.5 or urine output ≤ 500 ml/day.

SIRS (SYSTEMIC INFLAMMATORY RESPONSE SYNDROME)[32]

The American College of Chest Physicians (ACCP), Society of Critical Care Medicine (SCCM), The European Society of Intensive Care Medicine (ESICM), the American Thoracic Society (ATS) and the Surgical Infection Society (SIS) Diagnostic Criteria for SIRS 2001.

Diagnostic criteria for SIRS include two or more of the following:

Body temperature > 38° C or 36° C
Tachycardia with Heart rate > 90 beats per minute
Tachypnea manifested by respiratory rate > 20 breaths per minute or $PaCO_2 < 32$ mm Hg
Leukocytosis or white blood cell count > 12,000 cells/mm^3 or < 4,000 cells/mm^3 or significant immature neutrophils (>10%)
In 2001, the AACP, SCCM, ATS and ESICM updated the previous criteria to include the following:
Chills, decreased urine output, decreased skin perfusion, hypoglycemia
Poor capillary refill, petechiae, decreased platelet count, skin mottling
Unexplained changes in mental status

SEPSIS[11]

Sepsis is a change in the burn patient that triggers the concern for infection, the trigger includes at least three of the following: (it is age-dependent and adjustments are necessary for children)

I. Temperature > 39° or < 36.5° C
II. Progressive tachycardia
  A. Adults > 110 bpm
  B. Children > 2 SD above age-specific norms (85% age-adjusted maximum heart rate)
III. Progressive tachypnoea
  A. Adults > 25 bpm not ventilated
  B. Children > 2 SD above age-specific norms (85% age-adjusted maximum respiratory rate)
IV. Thrombocytopenia (will not apply until 3 days after initial resuscitation)
  A. Adults < 100,000/mcl
  B. Children < 2 SD below age-specific norms
V. Hyperglycemia (in the absence of pre-existing diabetes mellitus)
  A. Untreated plasma glucose > 200 mg/dl or equivalent mM/L
  B. Insulin resistance – examples include
    i. > 7 units of insulin/hour intravenous drip (adults)
    ii. Significant resistance to insulin (>25% increase in insulin requirements over 24 hours)
VI. Inability to continue enteral feedings > 24 hours
  A. Abdominal distention
  B. Enteral feeding intolerance (residual > 150 ml/hour in children or two times feeding rate in adults)
  C. Uncontrollable diarrhea (> 2500 ml/day for adults or > 400 ml/day in children)

In addition, it is required that a documented infection is identified

  A. Culture positive infection
  B. Pathologic tissue source identified
  C. Clinical response to antimicrobials

MODS (MULTIPLE ORGAN DYSFUNCTION SYNDROME)[33]

MODS is defined as (definition from ACCP and SCCM) "Presence of altered organ function in an acutely ill person such that homeostasis cannot be maintained without intervention. Primary MODS is the direct result of a well-defined insult in which organ dysfunction occurs early and (can) be directly attributable to the insult itself. Secondary MODS develops as a consequence of a host response and is identified within the context of SIRS (systemic inflammatory response syndrome)."

Etiopathology

The clinical response to major burns brings about a state of systemic inflammation. The initial insult due to burns on the soft tissues, the hemodynamic system and the presence of devitalized skin brings about an inflammatory change mediated by the biochemical mediators, which act both locally and systemically in defense of the host. A delayed or inadequate resuscitation predisposes the patient to MODS, also called 'Primary MODS'. During the course of major burns, there is a state of generalized systemic inflammation due to the presence of devitalized tissue, raw areas, inhalational injuries, split thickness donor sites, translocation of bacteria from the gut and release of inflammatory mediators from the hypoperfused gut, etc. Chen et al [34] reported that subeschar tissue fluid induced SIRS in healthy rats possibly due to production of excessive inflammatory mediators. The biochemical mediator response to these inflammatory stimuli has remote organ effects as well, beside the homeostatic and local defense it serves. The body is primed for any further assault. Inherent to the nature of burn trauma, burn wound infection is very likely and so are events like breakdown of the gut mucosal barrier, access port infection, etc. These repeated seemingly insignificant events in an environment of burn trauma induced systemic inflammatory state tend to maintain the internal milieu in a condition similar to SIRS. SIRS or Systemic Inflammatory Response Syndrome is a state of systemic inflammation due to varied reasons and is suspected when the body temperature, heart rate, respiratory rate and the leukocyte count are abnormal along with chills, decreased urine output, decreased skin perfusion, hypoglycemia, poor capillary refill, petechiae, skin mottling, decreased platelet count and unexplained mental aberration. In essence it is a reflection of the compromised state of the various organ systems. In certain circumstances this perpetual state of inflammation becomes uncontrolled and multiple organs of the body get affected. When this mechanism overwhelms the critical threshold, defective oxygenation occurs.[35] This results in remote dysfunction of one more organ not primarily affected by the burn trauma, most commonly the lungs. Multiple Organ Dysfunction Syndrome (MODS) is said to have set in when multiple organs are affected, the defining factor between SIRS and MODS being the inability of the host to use the available oxygen to its benefit in MODS. When this constant state of SIRS is perpetuated by the repeated stimuli and the host fails to activate the required counter-inflammatory measures, there is progressive deterioration in organ function and ultimately the death of the patient.[14]

Proposed by SCOR[23,36] (Specialized Center of Research) for MODS. Initial insult (1st hit) such as inadequate tissue perfusion, inflammation or dead tissue provoke the inflammatory system by "priming PMN" to a latter insult (2nd hit). The second hit can be any trauma, infection, stress or an episode of hypotension. Second hit is an altered response to injury and probably due to the initial activation of the first insult. This can be altered or delayed by inadequate resuscitation and is usually followed by an altered physiology like microcirculatory ischemia, deterioration of the gut barrier and if this sustained alteration continues it leads to sustained SIRS followed by MODS and death (Fig. 31.3).

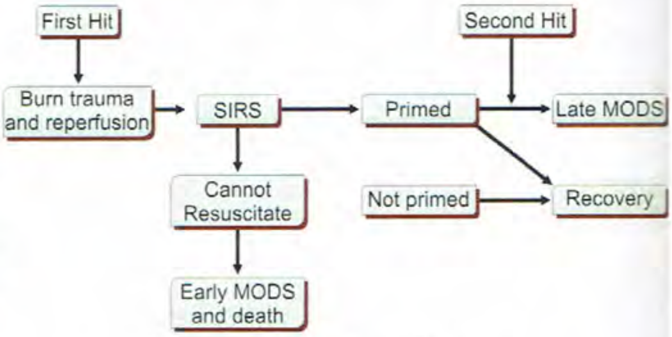


Fig. 31.3: Two hit model of MODS

Major burns are more likely to be complicated by MODS, which could set in at the beginning–*Primary MODS* or later in the course of treatment–*Secondary MODS*. In the large majority, the cause of death in critical burns is MODS. Hence, it is important to understand the pathogenesis of this condition and know the predisposing factors and conditions. The syndrome of multiple organ dysfunction is a systemic manifestation of the overwhelming inflammatory response incited by the trauma of burns or its consequent course of events. A list of major predisposing factors is mentioned below. These inciting factors or predisposing conditions may precipitate MODS on their own or act as small stimulating factors, which add up to aggravate the ongoing controlled inflammatory state to an unregulated one.

Predisposing Factors for MODS

Known risk factors for MODS in Burns are listed comprehensively as follows:

Advanced age
Inadequate resuscitation

Prolonged tissue hypoxia during resuscitation
Reperfusion injury following ischemia
Presence of devitalized/necrotic tissue
Chronic recurring infections/sepsis
Endotoxemia
Massive blood transfusion
Inhalational injury, aspiration, lung contusion
Renal insufficiency
Malnutrition
Diabetes, Cancer, Pancreatitis, Trauma, Immunosuppressant therapy, chronic alcoholism.

Pathogenesis[25,37-39]

The progression from SIRS to MODS has been extensively researched as regards the biochemical events and abundant facts have emerged. However, despite the abundance of experimental and clinical evidence, a single common underlying event or process as the reason for a patient to progress to MODS is yet to be precisely defined. It is difficult to state that MODS is a single pathological process with different expressions or a set of different pathological processes which have a set defined end result.[37] In spite of these limitations the different events leading to MODS need to be understood to be able to manage this condition better. The pathogenesis of MODS is being described as related to the more accepted theories.

Uncontrolled Infection

Sepsis in burns is a major cause for MODS. With extensive availability of necrotic tissue and compromised tissue vascularity, burn wound infection is the most expected source of sepsis. However, early wound excision and coverage with better infection control measures in burns have decreased the incidence of fatal sepsis and though the course of recovery is punctuated by multiple infectious episodes, autopsy studies do not show uncontrolled infection of the wound at the time of death from MODS.[17] Lungs and the gastrointestinal tract are also considered very likely sources for systemic sepsis. Sepsis from burn wound infection is a major cause of sustenance of the state of systemic inflammation and progression to MODS.

As such, though infection is a common association with MODS, it is not always present and nosocomial infection may be considered a manifestation of MODS rather than a cause of it.[40] Effective infection prophylaxis like SDD (selective digestive tract decontamination) has reduced the incidence of pneumonia, bacteremias, etc. but has not shown a significant reduction in mortality.[41-43]

It is a common understanding that it is the presence of uncontrolled infection that leads to MODS. However, currently it is theorized that infection usually follows MODS rather than preceding it. In fact, nosocomial infection is being considered a manifestation of MODS rather than a cause of it.[40] Nosocomial infection is uncommon in patients without organ dysfunction and its occurrence is directly proportional to the severity of MODS.[37] The causative organisms are usually *Candida*, Coagulase negative Staphylococci, *Pseudomonas* and Enterococci, which have low intrinsic virulence.

Endotoxin from the microbes may also be a reason for MODS and endotoxemia is more commonly seen than culture proven infection,[44,45] possibly due to absorption of endotoxins from the gut or lungs. Some of the bacterial products may have direct cytotoxicity on the basic cellular processes like induction of apoptosis of neutrophils or epithelial cells.

The unifying infectious etiology suggested gut as a potential source of bacteria (host for 10^8 aerobes and 10^{11} anaerobes in the colon) or at least circulating products of bacteria.[25] Hypoperfusion results in damage to the mucosal and immunological barriers, leading to translocation of endogenous bacteria or their products into the systemic circulation. This 'second hit' acts as a trigger for the precipitation of secondary MODS and distant organ effects especially to the lungs.[46] The hypoperfused mucosa is a source of inflammatory mediators as well.[15,47,48] Quantification of intramucosal pH (tonometry) can be used to stratify mortality risk.[49] Interestingly attempts to improve the gut perfusion have not changed the MODS outcome in septic patients.[45,50]

Uncontrolled Systemic Inflammation

More than the cytopathic effects of the microbes it is the uncontrolled inflammatory host response that is responsible for the progression of SIRS to MODS. The notable organ dysfunction remote from the site of injury or *functio laesa* of systemic inflammation is the result of the unregulated systemic inflammation, the severity of which is the determinant of the outcome of MODS.[51]

It is the activation of circulating cells (leukocytes), the endothelium, the liver and multiple mediator networks that are normally held in balance by the anti-inflammatory mediators.[25] The chemotactic agents attract, the adhesion molecules focus and the cytotoxic agents drive the inflammatory process and MODS occurs when the host's inflammatory or anti-inflammatory response (or both) to injury are excessive.[52] The myriad of actions from this panoply of chemical mediators push the patient either towards an inflammatory state or oppose it. It is the dysregulation of this balance that causes MODS.

The proinflammatory actions can be seen in the form of (1) Up regulation of the expression of inducible nitric oxide synthetase[53] resulting in increased release of nitric oxide and its resultant effects on microvascular resistance and capillary flow. (2) Augmentation of neutrophil cytotoxic mechanisms by inducing the release of oxygen radicals and proteolytic enzymes.[54,55] (3) Up regulation of endothelial cell adhesion molecule expression[56] (4) Activation of endothelial procoagulant activity.[57] (5) Inhibition of the expression of thrombomodulin,[58, 59] a critical factor in the activation of protein C anticoagulant pathway.

The other school of thought suggests the dysregulated inflammatory response in MODS to be due to a state of immunodeficiency or immune paralysis rather than excessive inflammation.[60] Such impaired immunity is suggested by (1) anergy to delayed hypersensitivity recall testing with common antigens;[61] (2) impaired *de novo* antibody synthesis to tetanus toxoid;[62] (3) reduced monocytes expression of human leukocyte antigen-DR (HLA-DR);[63] (4) increased circulating levels of counter-inflammatory cytokines such as IL-10[64] and transforming growth factor β.[65]

Raised circulating levels of cytokines such as IL-6 and TNF-α or markers of TNF-α released in response to the inflammatory stimuli are concomitant with organ dysfunction and high mortality.[66-70] Serum concentrations of these pleiotropopic mediators not corresponding directly to the mortality in MODS have also been reported.[71] However, epidemiologically, postoperative septic patients that have a high TNF concentration have a higher mortality and MODS than those with low TNF levels.[70]

Huang et al in a prospective study and previous studies[71-79] reported that post burns induction of SIRS was due to the chemical mediators, which were mainly injurious to the endothelial cells. Early eschar excision decreased SIRS and prevented organ dysfunction and MODS. As part of the study they observed deformation of the endothelial cell when it was exposed to the sera of patients undergoing staged escharotomy compared to no deformation of the endothelial cells when it was exposed to sera of postoperative patients of early eschar excision.

Tissue Hypoxia

Cellular hypoxia seems to be a very plausible final common pathway for organ failure due to the diverse etiologies.[80-81] It is either due to reduced oxygen delivery or oxygen utilization leading to the cellular physiologic derangements. Adequate resuscitation restores the oxygen deficiency on the whole but regional hypoxia to the tissues, especially to the gastrointestinal tract[82,83] or brain[84] is known to occur. Meaningful resuscitation, thus, aims to correct systemic and regional tissue hypoxia as soon as possible. It has been seen that survivors have a higher cardiac index, lower systemic vascular resistance and higher oxygen consumption than non-survivors.[85] Cardiac index more than 4.5 L/min/m^2, oxygen delivery index greater than 600 ml/min/m^2 and oxygen consumption greater than 170 ml/min/m^2 are critical.[86] Tissue hypoxia, global or regional, is reflected biochemically as increased concentrations of lactate and correlates with adverse prognosis.[87,88] Methods to improve oxygen delivery by transfusions[89] or large doses of dobutamine[90,91] were thought to be very effective but recent trials do not support it. In fact they may worsen the outcome and increase the severity of organ failure. Derangements in the intracellular utilization of oxygen in the presence of adequate oxygen delivery are the likely reason of tissue hypoxia or 'cytopathic hypoxia'.[92]

Tissue ischemia due to hypoperfusion results in depletion of energy stores and accumulation of tissue metabolites. In this scenario, resumption of tissue perfusion is crucial. However, reperfusion can result in more tissue damage through O_2 derived free radicals, O_2, H_2O_2 and OH. Reactive oxygen species (ROS) are formed immediately, lasting for 2 to 5 minutes. These can damage lipid membranes, enzymes, nucleic acid and receptors eventually leading to cell death. Degeneration of the nucleic acid by Xanthine oxidase can generate O_2 and H_2O_2 during the oxidation of hypoxanthine and xanthine, logically the enzyme inhibitors can be protective. The neutrophils are abundant in splanchnic vessels and their recruitment occurs during ischemia and reperfusion. Neutrophils contain NADPH which reduces O_2 to free O_2 radicals and myeloperoxidase in neutrophils forms HOCL from Cl and H_2O_2 ions. HOCL is 100 times more reactive than H_2O_2 as an oxidizing agent and mediates cell damage. The oxygen free radicals have the following effects (1) alteration of the vascular permeability, (2) cell lipid peroxidation altering function, (3) initiation and perpetuation of local and systemic inflammation, (4) disruption of interstitial matrix, (5) impairment of macrophage phagocytic ability, (6) alteration of cell DNA and (7) initiation of arachidonic acid metabolism.

Dysregulated Apoptosis

Apoptosis or programmed cell death is a process by which cells activate an endogenous program that leads to controlled cell death and its transformation to membrane bound vesicles that are cleared by macrophages without inciting an inflammatory reaction.[93] This is a basic step in the embryonic development, immune maturation, aging, cell turnover at epithelial surfaces and resolution of inflammation. Dysregulated apoptosis or premature cell death is one of the pathways for organ dysfunction in critical illness. MODS –associated release of cytokines, interleukin-1, interleukin-6, NF heat shock proteins, oxidants and glucocorticoids alter the rate of tissue apoptosis.[37,94] It has been drawn in as a reason for liver, kidney and cardiac disease.[95-97] On the contrary, apoptosis may be necessary for the resolution of inflammation. Hence, the timing of apoptosis and the cells involved are the main determinants of whether the apoptosis will be favorable or unfavorable for the patient.[37] Studies have suggested that mitochondrial dysfunction resulting in the crucial cytopathic hypoxia or apoptosis is a key factor in the severity and duration of MODS.[98] Methods to modulate the expression of apoptosis are not available clinically and hence their role is unproven.[37]

Microvascular Coagulopathy

The expression of inflammation and that of coagulopathy are regulated by mechanisms which are interlinked and hence, it is likely that with the activation of inflammation there could be concomitant alteration in microvascular coagulation during states of systemic inflammation. Many studies suggest a crucial role of inappropriate intravascular coagulation as a final common pathway to organ dysfunction. It is known that disseminated intravascular coagulation is a powerful predictor of MODS and platelet counts below 80,000/ml has a sensitivity of 83.3 % and a specificity of 100 % for the prediction of MODS.[37] A shift towards the procoagulant state commonly occurs in the critically ill[37] and this shift usually precedes the development of organ dysfunction[99] and persists in those who go further downstream and die.[100]

The process of coagulation can be stimulated by tissue factor on endothelial cells and monocytes. Besides the physiological stimulus, this process could be induced by endotoxin,[101] cell surface components of Bacteroides fragilis,[102] by inflammatory cytokines[58,103] or by integrin cross linking.[105] The coagulation cascade culminates in cleavage of fibrinogen to fibrin and fibrin deposition, which besides the physiological role of hemostasis and localization of micro-organisms, obstructs oxygen delivery and further aggravates inflammatory injury by hypoxia or through signals delivered to the thrombin receptor. A number of proinflammatory gene products are released with the activation of the thrombin receptor and it results in the release of nitric oxide.[105]

The biological regulators of coagulation are Antithrombin III, Tissue factor pathway inhibitor and Protein C; they have different mechanisms to contain the process of coagulation. Protein C is a serine protease that complexes with protein S to inhibit factors Va and VIIIa,[106] circulates as an inactive precursor and is synthesized primarily in the liver.[107] It is activated by the binding of thrombin to thrombomodulin on the endothelial surface. Protein C binds to a specific receptor on endothelial cells, the endothelial cell protein C receptor (ECPR), this increases the release of protein C by thrombin-thrombomodulin complexes.[108] Activated protein C (APC) interactions are predominantly anti-inflammatory, conversely inflammation reduces APC levels and activity.[37] Further to these complex interactions, apoptosis and coagulopathy are interlinked through the expression of phosphatidylserine.

A change to a procoagulant state is usually seen in the critically ill patients and the increase in procoagulant markers (Table 31.1) precedes the development of organ dysfunction[99] and persists in those who develop MODS or die.[100] Protein C levels are found to be decreased in acute renal failure[109] and in patients with neutropenia.[110]

Logically, modulation of the mediators regulating coagulation looks possible and since dysregulated coagulation seems to be the crux for the development of MODS, attempts have been made to target the coagulation cascade and have shown increased survival in experimental models.[111-118] Convincing results from human trials showing improvement in clinical results is awaited. A successful intervention at this level has shown promising results in clinical trials and hopefully would translate into effective therapy.

It has been found that the autonomic nervous system continuously communicates amongst all vital organs in the normal functioning of the body and an autonomic dysfunction can result in severe inflammation and SIRS, which could progress to MODS.[122]

Table 31.1: Coagulation abnormalities in trauma and sepsis

Increased	*Decreased*
Procoagulant activity/Tissue factor	Platelets
Fibrinogen	Antithrombin III
Thrombin-antithrombin	Protein C antigen
Fibrinopeptide A	Protein C inhibitor
Prothrombin fragment 1 + 2	Protein S
Plasmin alpha-2-antitrypsin complexes	Factor VII
D-dimers	Factor XII
Thrombomodulin	
tPA activity	
tPA antigen	
Plasminogen activator inhibitor-1	
von Willebrand factor	
von Willebrand factor propeptide	
Platelet thrombospondin	

The number of theories (Table 31.2) explaining the basis of MODS betrays the fact that its nuances are still not fully understood, especially the mechanism of multiple organ failure. However, collectively many of the observations have been explained through these theories, which form the basis of the interventions that have shown promising results in both the experimental and clinical setup.

Scoring

Though the understanding of this recently uncovered condition, MODS, is not crisp and clear but the management of this critical illness demands an accurate and detailed day to day assessment. Since the key to win the battle against MODS is its prevention, it is imperative that any downstream events in the course of treatment are detected early, when it is likely to be controlled before it becomes autonomous. A number of clinical measurement systems have been tried for the evaluation and monitoring of MODS in critical care patients. The basic clinical need for a monitoring system is to be able to document the course of events, the status of the organs involved, whether the damage is reversible or not and hopefully predict the outcome with a scope for gradation of the dysfunction regarding its severity rather than just a dichotomous data.

For easy clinical assessment and reproduction of data, the parameters and the conditions need to be defined. A number of definitions and parameters are mentioned as a yardstick for a standard monitoring of the patient. Most of these conditions have been defined on the basis of research on non-burn critically ill subjects; hence some of the definitions do not appear logical for patients of burns progressing from SIRS to MODS and death. For instance SIRS or the state of systemic inflammation is normally present in large burns due to the hypermetabolic state. The baseline temperature is reset to 38.5°C, tachycardia and tachypnea persist for months in patients with extensive burns and continuous exposure leads to leucocytosis. Hence, the usual criteria for SIRS are normally present and confounding if they were to be taken as markers for a deteriorating condition.

The recommendations of the American Burn Association Consensus Conference to define Sepsis and Infection in Burns in January 2007[11] are very pertinent in this matter. They are as follows:

Table 31.2: Conceptual models of MODS and the therapeutic aim[37]

Pathologic process	*Manifestation*	*Therapeutic implications*
Uncontrolled infection	Persistent infection, nosocomial-ICU acquired infection, endotoxemia	Aggressive use of antibiotics and source control measures
Systemic inflammation	Cytokinemia (particularly IL-6, IL-8, TNF), leukocytosis, increased capillary permeability	Neutralization of specific cytokines (IL-1, TNF, PAF) or of activational pathways
Immune paralysis	Nosocomial infection, increased anti-inflammatory cytokine (IL-10) levels, decreased HLA-DR expression	Granulocyte Colony Stimulating factor, Interferon
Tissue hypoxia	Increased lactate	Augmentation of DO_2
Microvascular coagulopathy and endothelial activation	Increased procoagulant activity, decreased anticoagulant activity, increased von Willebrand factor, soluble thrombomodulin, increased capillary permeability	Augmentation of anticoagulant mechanisms (APC, TFPI, Antithrombin)
Dysregulated apoptosis	Increased epithelial and lymphoid apoptosis, decreased neutrophil apoptosis	Caspase inhibition
Gut-liver axis	Increased infection with gut organisms, endotoxemia, Kupffer cell activation	Selective digestive tract decontamination, enteral feeding

APC- activated protein C, IL-interleukin, PAF - platelet activating factor, TFPI – tissue factor pathway inhibitor, TNF – tumor necrosis factor

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS)

SIRS should not be applied to burns. Though patients of moderate to major burns have not been evaluated systematically, they normally exhibit tachypnea and tachycardia with raised body temperature. Since no studies of the epidemiology and time-course manifestations of SIRS in burns have been done, it is less meaningful to prognosticate the course of burns with the criteria for SIRS. Normally, persistence of SIRS for more than three days after trauma was associated with increased risk of MODS and death. However, burn trauma is different in having persistence of signs of SIRS even after the acute inflammation has subsided and its persistence does not necessarily suggest any clinical complication or sepsis. And no study has demonstrated that at a certain point in the course of burns the SIRS criteria becomes significant. Moreover, the course of burns can be fairly well estimated by the surface area of burns, age of the patient and co-morbidities while SIRS or its related scoring systems like APACHE II, etc. fail to gauge the severity of the situation. It has been suggested that increasing the temperature component of SIRS to 38.3 would increase its specificity.[120] Besides having been developed for adults, SIRS is not helpful for clinical trials.

The ongoing inflammation could be defined by the presence of base deficit and/or lactic acidosis and it correlates with the severity of injury and mortality in burn and trauma patients.[121-123] It remains to be seen whether the absolute values or their persistence over time is more important. Other biochemical markers of significance are C reactive protein,[124] procalcitonin,[125] tumor necrosis factor-alpha,[126] interleukin-6, etc.[127-130] They, however, do not appear to correlate adequately with the prognosis in burn patients.[131]

Once again these changes in clinical parameters may be normally present in a burnt patient without any overt infection. Hence, the definition of burn sepsis must differentiate changes in the patient's status due to microbial infection from changes due to burns itself or its associated complications like inhalation injury. Due to the hypermetabolic state the baseline temperature is reset to 38.5°C in patients with large burns. Hence, fevers are not considered a sign of sepsis unless they reach at least 39°C. Chronic wound exposure due to open burns or split thickness donor sites induces leucocytosis on its own and hence, an unreliable sign of infection. However, thrombocytopenia after 3 days of resuscitation is a valuable sign of sepsis.

The MODS scoring systems can be classified using general physiological critical care scores (Acute Physiological and Chronic Health Evaluation – APACHE, Simplified Acquired Physiological Score – SAPS, Mortality Probability Model – MPM[132] or specific organ dysfunction scores (Multiple Organ Dysfunction Score – MODS, Sepsis Organ Failure Assessment – SOFA (Table 31.4), Logistic Organ Dysfunction System – LODS). The inference from scoring systems could be dichotomous, suggesting an organ failure to be present or not[133] and some provide information regarding the severity (graded)[134-136] of organ dysfunction in which a deterioration creates a higher score. The different scoring systems may use different criteria or different organs or be different in assessment in the point of time during the course of the disease. The American Burn Association consensus conference in January 2007[11] made the following recommendations regarding scoring systems for MODS in burns.

- The committee decided to use the Marshall MODS Scoring System (as modified by Cook) till a better system was found (Table 31.3).
- The ACCP/SCCM consensus criteria for sepsis should not be used, especially during the initial period of volume resuscitation.
- Organ dysfunction/failure should not be assessed until the acute resuscitation period is over (approximately day 3 post burn). Since major burns require large fluid

Table 31.3: Modified Marshall MODS Scoring System [137]

Organ System	*0*	*1*	*2*	*3*	*4*
Cardiovascular (heart rate, inotropes, lactate)	≤120	120-140	>140	Inotropes	Lactate >5
Respiratory, PO_2/FIO_2	>300	226-300	151-225	76-150	≤75
Renal (creatinine, µmol/L)	≤100	101-200	201-350	351-500	>500
Central nervous system (Glasgow Coma Score)	15	13-14	10-12	7-9	≤6
Hepatic (total bilirubin, µmol/L)	≤ 20	21-60	61-120	121-240	>240
Hematologic (platelet count × 10^3)	>120	81-120	51-80	21-50	≤20

Six domains of the MODS. The original MODS cardiovascular component was defined by the heart rate X right atrial pressure/mean arterial pressure. The modified cardiovascular component of MODS is defined as follows: 0, heart rate <120 beats per minute (bpm); 1, heart rate 120-140 bpm;2, heart rate >140 bpm; 3, need for inotropes more than dopamine >3 µg/kg/min; 4, serum lactate >5 mmol/L. The scores can range between 0 and 24.

transfusions during resuscitation there is a massive fluid shift in the vascular and extravascular space with significant reversible changes in the levels of important parameters like leukocyte and platelet count, which reflect a temporary decrease due to the dilution effect.

- Organ dysfunction scores should preferably reflect the severity of organ failure rather than just the presence or absence of it (Table 31.5). It would conceptually be an advantage to start therapeutic action early in response to a scoring system that would reveal deterioration in organ functions rather than simply presence or absence of failure. The scoring systems that grade the organ dysfunction are the Marshall's MODS scoring system, Cook's modification of the Marshall MODS scoring system, SOFA (Sepsis – related organ failure assessment), the Brussels score and LODS (Logistic organ dysfunction system). These are specific organ scoring systems in contrast to the scoring systems using the general physiological critical care scores like APACHE (Acute Physiological and Chronic Health Evaluation), Simplified Acquired Physiological Score (SAPS), Mortality Probability Model (MPM), etc.[134-136] Amongst SOFA and the modified version of Marshall's MODS scoring system, the later has been used in burn

Table 31.4: Sequential Organ Failure Assessment (SOFA) score[133]

Organ failure/Variable	*Score 0*	*Score 1*	*Score 2*	*Score 3*	*Score 4*
Respiratory PaO$_2$/FiO$_2$, mmHg	≥ 400	< 400	< 300	< 200 on MV	< 100 on MV
Hematology Platelets, 10^9/L	≥ 150	< 150	< 100	< 50	< 20
Liver Bilirubin, mg/dL	< 1.2	1.2-1.9	2.0-5.9	6.0-11.9	> 12.0
Cardiovascular					
Mean arterial blood pressure, mmHg	≥ 70	< 70			
Dopamine, µg/kg/min			≤ 5	> 5	> 15
Dobutamine, µg/kg/min			any dose		
Epinephrine, µg/kg/min				≤ 0.1	≤ 0.1
Norepinephrine, µg/kg/min				≤ 0.1	≤ 0.1
Central Nervous System Glasgow Coma Scale	15	13-14	10-12	6-9	< 6
Renal					
Creatinine, mg/dL	< 1.2	1.2-1.9	2.0-3.4	3.5-4.9	≥ 5.0
Urine output, mL/day	≥ 500			< 500	< 200

Table 31.5: Parameters and scores used in assessing organ dysfunction[25,134-136]

Parameter	*SOFA*	*Score*	*MODS*	*Score*	*LODS*	*Score*
Respiratory	PaO$_2$/FiO$_2$ and ventilation	0 to 4	PaO$_2$/FiO$_2$	0 to 4	PaO$_2$/FiO$_2$ Ventilation and CPAP	0-3
Coagulation	Platelet number Cell number	0 to 4	Platelet number	0 to 4	Platelet, white blood	0-3
Hepatic	Bilirubin	0 to 4	Bilirubin	0 to 4	Bilirubin, Prothrombin time	0-3
Cardiovascular	Blood pressure and vasopressor use	0 to 4	Heart rate, Central venous pressure, blood pressure	0 to 4	Heart rate and blood pressure	0-3
Central Nervous System	Glasgow Coma Scale	0 to 4	Glasgow Coma Scale	0 to 4	Glasgow Coma Scale	0-3
Renal	Creatinine Urine output	0 to 4	Creatinine Urine output	0 to 4	Creatinine Urine output	0-3
Aggregate score	Add worst daily scores	0 to 24	Add worst daily scores	0 to 24	Add worst daily scores	0-22

patients[138,139] and the parameters are easily obtained clinically. The neurological assessment is subjective in both the MODS and SOFA scoring systems and may affect the overall accuracy.

The modified Marshall MODS scoring system and SOFA scoring system are useful for a pre-emptive diagnosis of organ dysfunction during the management of critical burn patients. A daily worst score is useful to detect any deterioration and is likely to improve the outcome by facilitating an early therapeutic intervention.

MANAGEMENT OF MODS

The management of MODS in burns encompasses a few salient principles, prevention of MODS being the ultimate goal because once a certain stage of systemic inflammation is reached, set protocols of management fail to make a significant effect, treatment becomes empirical and prognosis poor. The spectrum of activity of the numerous pleiotropopic inflammatory mediators affects almost every system of the body with proinflammatory and anti-inflammatory actions closely enmeshed; making the choice of the time and mode of therapeutic intervention at the molecular level very difficult and risky. Hence, despite the advancements in experimental and clinical research, the basis of treatment of MODS remains primarily its prevention and alleviation of inflammatory stimuli and organ support when the scoring parameters suggest progression towards organ dysfunction. A brief outline about management strategy is discussed.

The management of MODS in burns aims at

1. Prevention
2. Identification of risk factors
3. Control inflammatory stimuli
4. Balance oxygen supply and demand
5. Nutrition
6. Specific organ support
7. Specific therapeutic interventions

Prevention

The prevention of multi-organ failure is likely to be more effective than its treatment – Baue.[19] Besides adequate timely resuscitation of burns and treatment of other attendant injuries and co-morbidities, a proactive approach for timely detection and management of any event that would potentially stimulate the inflammatory state would likely contain the SIRS from progressing to MODS. It is important to understand that all the preventive actions need to be early in the course of any event as it unlikely to have a significant impact once it becomes autonomous.[37] For example, prevention of infection in critical illness may reduce the morbidity and mortality[43] but once the proinflammatory cascade is activated their perpetuation may not require the presence of infectious stimuli. Hence, the late treatment of infection may not ameliorate its effects and prevent the clinical deterioration of the patient. The neutralization of the proinflammatory mediator release in patients with sepsis has not shown a significant improvement in outcome.[44] On the contrary, coagulopathy develops late in the cascade of the inflammatory response and therapeutic intervention against it appears more achievable.[37]

Some of the accepted therapeutic options that aid in prevention of the inflammatory state from progressing to MODS are:

1. Adequate timely fluid resuscitation and early homeostatic normalcy.
2. Early burn wound excision and coverage – besides decreasing chances of sepsis and inflammation, it also decreases release of inflammatory mediators.
3. Preservation of gut mucosal barrier by maintaining adequate perfusion and enteral feeding.
4. Efficient management of inhalational injuries.
5. Management of the pre-existing co-morbid conditions like diabetes, cardiac disease, etc.
6. Maintaining homeostasis and peripheral oxygen delivery.

High degree of suspicion for infection of the burn wound and port access sites is required as typical physical signs may not be well distinguished in this state of systemic inflammation. Hence it is important to look for the risk factors.

Identification of Risk Factors

Besides the above known threats for instigation of MODS, any other vulnerable inciting factors should be contained. It is important to be constantly vigilant with the "two hit theory" of MODS[7] in context, where the onset of secondary MODS is more likely even by small adverse stimuli as the immune system is already primed by the initial insult on the inflammatory system. Though each small inciting stimulus on its own would not precipitate MODS but a constant flux of multiple such events may tip the course of major burns towards MODS and eventual death in a large majority.

Patients of burns are prone to unusual and often occult infections.[140] The important risk factors are infection at the port access sites (intravenous access, urinary catheters, central lines etc). The standard protocols for infection control have a very important role. Suppurative thrombophlebitis and endocarditis are known to occur without localizing signs.[38] Ventilator associated respiratory infection and pneumonia is a known hazard of assisted ventilation and suppurative sinusitis in those with nasotracheal intubation.[141] Burn patients are more susceptible to pneumonia[142,143] because of (1) relative immunosuppression (2) dysfunctional ciliary movement in inhalational injuries leading to impaired secretory clearance (3) pulmonary inflammatory activation with acute lung injury (4) increased leakage of nutrient rich plasma into the lung parenchyma (5) ventilator associated injury due to aggressive ventilation because of decreased lung and chest wall compliance. Acalculous cholecystitis may present with generalized sepsis without any localizing signs.[38] All these unusual complications of burns are managed as per the specific requirement and protocol.

Control of Inflammatory Stimuli

Early excision of devitalized tissue and coverage of raw areas as far as possible is the key to prevention of infection in burns and the ensuing inflammatory state. The uncovered wound and the devitalized tissue are also a source of stimulus for release of the biochemical mediators of inflammation, such as interleukin-6, interleukin-8, tumor necrosis factor, etc. These mediators besides causing a hypermetabolic and inflammatory state, can have remote organ effects, for instance interleukin-8 from the wound gets upregulated in the lungs and is responsible for pulmonary dysfunction.[38] Even split thickness donor sites invoke an inflammatory response. On the other hand, complete wound closure without donor sites decreases oxygen consumption and the inflammatory response from the wound. Huang et al[144] reported early eschar excision to be effective in preventing and treating early post burn organ dysfunction and MODS, mainly by alleviating SIRS and endothelial cell injury.

Wound sepsis has been a major cause of the persistent inflammatory state and MODS. It is however, decreasing in incidence due to early wound coverage and these days death from infection is more commonly due to pneumonia than due to wound sepsis.[38] Burn patients who are at risk of MODS should be closely monitored for any signs of infection and treatment promptly instituted. Standard infection control protocols need to be strictly followed to curb any stimulation of the inflammatory state. Though the institution of anti-inflammatory therapy with ibuprofen[145] or corticosteroids seemed to be an attractive option but have not been found to be useful in SIRS or MODS.[146,147] On the contrary, the immunosuppressive effect of steroids predisposes to complications.[146,148,149] Experimental studies indicate that granulocyte/macrophage colony stimulating factors may enhance the compromised immune system and be useful.[150] It has been found useful for chronic neutropenia and in bone marrow transplantation.

Theoretically, MODS therapy directed against inflammation are:[25] (1) cell adhesion retardation; (2) inflammatory mediator reduction (translation/transcription inhibition)(3) neutralizing (polyclonal or monoclonal) antibodies directed at cytokine/vasoactive/coagulation/complement mediators; (4) cytokine/vasoactive/coagulation/complement mediator receptor inhibitors; (5) anti-inflammatory protein induction (preconditioning, substrates, products, or genes); and (6) anti-oxidants and anti-proteases.[151] The newer developments entail measurement of specific elements[152] in the inflammatory cascade and countering the abnormally increased ones with cloned proteins and antibodies (monoclonal and polyclonal).[153] Thus, it envisioned to continuously titrate and adjust anti-inflammatory therapy in response to specific biochemical changes in the inflammatory cascade.[154,155]

Balancing Oxygen Supply and Demand

Cellular hypoxia, due to either decreased supply (impeded extraction of oxygen as in sepsis) or increased demand changes the ATP generation in the cells from the aerobic to the anaerobic pathway. It results in a rise in intracellular hydrogen ion and calcium with a decrease in function of the sodium calcium pump.[38] It is also responsible for the release of oxygen free radicals. When all these persist due to persistent low oxygen tension, the membrane phospholipids maybe degraded and result in cellular dysfunction. Clinical data suggests that inadequate oxygen delivery is associated with MODS.[38]

Knowing the amount of oxygen being consumed by the patient is more valuable than oxygen saturation and cardiac output. It helps optimize the oxygen supply, reverse any oxygen debt and makes it easier to understand which interventions help decrease oxygen demand. It is the basis of maintaining a balance of oxygen supply and demand. Survival rates have been shown to improve where supra-normal values of cardiac index, oxygen delivery and consumption were reached.[85,156]

Normally the (VO_2) consumption is dependent on (DO_2) up to a certain point called critical point. Beyond this point

VO_2 will not increase no matter how much DO_2 is increased. The increasing slope becomes a plateau (Fig. 31.4). In septic shock, hypoxia and MODS, this critical point increases and in order to maintain the same VO_2, supra normal levels of DO_2 have to be provided. The adequacy of VO_2 can be monitored by the fall in lactic acid level, increase in mixed venous saturation (>75%) and improvement of gastric mucosal pH (N=7.34) and tissue oximetry.

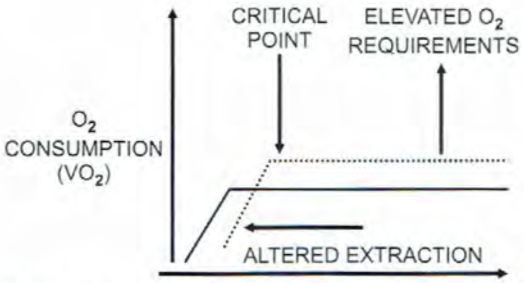


Fig. 31.4: Elevated and altered oxygen requirement in MODS

Decreasing Oxygen Demand

Since the oxygen demand is more than the supply (e.g. deficient peripheral oxygen extraction in sepsis) in MODS, it is logical to increase the supply and decrease the demand of oxygen for a favorable outcome. In fact interventions to reduce the oxygen demand are equally valuable as increasing oxygen supply. Fever, chills, tachycardia, tachypnea and pain are some of the basic clinical conditions, which if controlled could reduce the oxygen demand on an already deficient system. Assisted ventilation, hypothermic devices, pain medications, sedation, analgesics are elementary but effective in reducing the oxygen demand.

Specific Organ Support

Respiratory

Maintenance of adequate airway patency by intubation and supplementary O_2 therapy may have to be done early. If the patient is refractory, he/she should be put on mechanical ventilation. Normally a tidal volume–10-15ml/kg, FiO_2 -1/L with PEEP of 3-5 cm of water is sufficient to avoid pulmonary edema. To avoid O_2 toxicity PEEP of 10-15 cm of water with end inspiratory plateau pressure of 30-35 cm are sufficient. Low tidal volume ventilation, allowing permissive hypercapnia has shown to reduce ventilator associated lung injury and significantly improves the outcome.[157] Alternative ventilatory strategies are high frequency oscillatory ventilation and high frequency percussive ventilation.[158] There is increased risk of ventilator associated lung injury in burns due to the reduced pulmonary compliance and chest wall rigidity, which may lead to aggressive ventilatory management and high airway pressures.[157] If the oxygenation is still not adequate, then attention should be paid to cardiac output and hematocrit to optimize O_2 delivery. Extra corporal membrane oxygenator (ECMO), Intravenous oxygenator (IVO), surfactant therapy, and partial liquid ventilation with perflourocarbon are the ultimate salvage options.

Adjunctive therapies include inhaled nitric oxide for hypoxic vasoconstriction thereby improving ventilation/ perfusion mismatch and tissue oxygenation.[159,160] Aerosolized heparin and N-acetylcysteine have shown promising results.[161,162]

Cardiovascular System

Adequate resuscitation for homeostatic stabilization with end points being return of parameters to normalcy. Lactate levels are an important means of ascertaining the adequacy of tissue oxygenation. Myocardial function is depressed as a part of global ischemia and circulatory myocardial depressant substance. If this fails to reverse the hypotension and tissue perfusion, inotropes and vasopressors may be used. Dopamine 1-5µu/kg/min exerts β agonist dopaminergic effects. Low dosage Dobutamine is preferred for vasodilatation. It also increases O_2 delivery. Noradrenaline, ephedrine, phenyl ephedrine can be used to maintain the blood pressure. In the initial phase Dopexamine dilates splanchnic vessels when gut ischemia is suspected. The supports should be weaned as soon as possible. Maintaining hemoglobin concentrations at 7.0-9.0 g/dL may be superior to maintaining it at 10.0 to 12.0 g/dL in critically ill patients except those with acute myocardial infarction and unstable angina.[89] In fact, a recent study reported an increased mortality in burn patients associated with blood transfusions.[163] In a trend to avoid blood transfusions, recombinant human erythropoietin was tried but did not show any significant improvement in post burn anemia. However, the animal study showed improved healing of the burn wound.

Hematological

DIC and thrombocytopenia is a common feature in sepsis and MODS. As a part of therapy, Vitamin K and FFP are helpful in providing clotting factors.[22] PRP, platelet concentrate and fresh blood should be used in anemia with platelet counts < 50,000/mm^3. 1-Deamino-8-arginine vasopressin (DDAVP) in the dose of 0.3 mg/kg in 50 ml of saline is used in platelet dysfunction due to uremia.

Renal and Hepatic

It is essential to avoid all nephrotoxic and hepatotoxic drugs. Quick, adequate resuscitation can provide adequate perfusion to these important organs for them to function normally. Mannitol and frusemide may be needed to prevent hepatorenal shutdown. Use of neomycin for decontamination of gut and use of lactulose can avoid hepatic encephalopathy. If renal functions are not improved, patient may need dialysis.

Nutrition

Major burns are associated with a catabolic state with a significant increase in calorie requirement, which if not supplemented from outside, uses the available resources in the body leading to muscle protein catabolism, immune dysfunction, etc. The use of early enteral feeding may attenuate this catabolic response after thermal injury.[164] Calorie supplementation by enteral or parenteral (not preferred) feeding has been known to reverse the muscle catabolism. However, an aggressive high calorie feeding has been found to be associated with increase in mortality.[165] It is now recommended to provide adequate calorie intake via the enteral route with avoidance of over feeding. A high carbohydrate and high fat content in the diet may balance the skeletal muscle protein.[166] Understandably, an aggressive monitoring and treatment of hyperglycemia is highly recommended.[167,168]

Specific Therapeutic Interventions

Modulation of Hormonal and Endocrine Response

Burn trauma is associated with a state of catabolism and raised levels of catecholamines. Beta-adrenergic blockers have been studied and have shown some encouraging results.

The use of Beta-adrenergic receptor blockers in the patients of burns have been found to blunt the effects of catecholamines by attenuating hypermetabolism, decreasing oxygen demand and resting energy expenditure and decreasing heart rate and cardiac oxygen demand.[169] It has also shown decrease in catecholamine induced muscle catabolism and lipolysis.[170, 171] The catecholamine mediated defect in lymphocyte activation is modified by the beta blockade and hence, improves the immune response with decreased infectious complications.[172] Clinical studies have shown its usefulness in both adults and the pediatric population[173-176] with decreased mortality and wound infection rate and improved wound healing time. Further clinical trials are needed to authenticate its usefulness.

Immunotherapy

Immune modulation may be of use in targeting the systemic inflammatory response, infection, sepsis and immunosuppression.

Immunonutrition: Immune enhancing diets/agents have been studied for patients with extensive burns, which have a high risk of infection, sepsis and immunosuppression leading to an increased risk of organ dysfunction.[177] These diets/agents contain arginine, glutamine, omega-3 fatty acids and anti-oxidants such as ascorbic acid (Vitamin C) and α-tocopherol (Vitamin E). A combination of these constituents is tried and has been investigated. Glutamine has been shown to have very favorable results. However, the trials and multiple studies have agreed that indiscriminate use of the combination formula is not beneficial and a selective approach is proposed.[178-182] Anti-oxidants such as ascorbic acid (Vitamin C) and α-tocopherol (Vitamin E) have been shown to reduce the morbidity and prevalence of organ failure in the critically ill.[183, 184]

Recombinant Human Activated Protein C:[177] Recombinant activated protein C (activated drotrecogin alfa) is the first agent approved by the Food and Drug Administration for the treatment of severe sepsis. The Protein C Worldwide Evaluation in Severe Sepsis (PROWESS) trial has shown a statistically significant relative risk reduction of 28 day mortality.[118] Other trials have also shown a favorable result. However, there have not been any published trials on burn patients. Based on the available information, activated protein C can be considered for those in severe sepsis, who lack the exclusion criteria. The exclusion criteria includes within 12 hours of surgery, pregnancy, recent cerebrovascular accident, intracranial pathology, active bleeding or anticipated need for surgery within a 96-hour period.

Topical immunomodulation:[177] Burns induces local dermal inflammatory response, which in the absence of inhalational injury, is the source triggering the systemic inflammatory response by the release of a vast spectrum of proinflammatory mediators. Experimentally it has been seen that control of the dermal inflammation by topical application of p38-mitogen-activated protein kinase inhibitor to the burn wound abates the systemic inflammatory response and acute lung injury.[185] However, this has yet to be proved clinically.

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Chapter 32

Guidelines for Nutritional Management of the Burn Patient

Savita Arora

INTRODUCTION

Aggressive nutritional support in severe burns is an essential life saving therapy as important as control of infection and early excision and skin grafting. Due to the presence of acute phase responses in starving critically ill patients, a number of pathophysiological alterations develop caused by severe metabolic, hormonal, immunologic and circulatory disturbances. They lead to extensive nitrogen loss, malnutrition, markedly increased metabolic rate and immunologic deficiency predisposing the burn patients to frequent infections, poor wound healing, increased length of hospitalization and increased mortality.

Driven by the series of inflammatory mediators, catecholamines and hormones, the catabolic state leads to increased nitrogen loss due to increased amino acid efflux from the skeletal muscles, in order to accommodate increased demand of amino acids for tissue repair, acute phase protein production, cellular immunity and gluconeogenesis. This leads to diminished lean body mass which directly relates to outcome. Treatment of burn patient is incomplete and often unsuccessful if proper nutritional care is not provided. By way of nutritional management, it is possible to modulate the inflammatory response, immune status and promote burn wound healing.

Nutritional support requires high protein and high energy diets to provide metabolic support during heightened inflammatory state by giving sufficient calories and protein to minimize further loss of lean body mass (LBM). A 40% loss in body weight or a loss of one half to one third of protein is associated with death, particularly if this is rapid, as it may be in extensive burns.

Wound healing is limited when weight loss exceeds >20% of the pre-burn values. 10% weight loss is acceptable.

Oral feeding is the preferred method of providing nutrition to a patient with small burns. In extensive burns, however, nutritional therapy requires careful decision-making regarding the safe use of enteral and parenteral nutrition and the aggressiveness of nutrient therapy.

The effects of increased dietary components such as glutamine, arginine and omega-3 fatty acids and related compounds have been evaluated in burn victims. The components, when supplied in quantities two to seven times of those in normal diets of healthy persons, appear to have beneficial pharmacological effects on the pathophysiological alterations associated with burns.

PHYSIOLOGIC AND METABOLIC RESPONSES TO BURN INJURY

Effective nutritional therapy in burn patients involves an understanding of the physiologic and metabolic alterations that accompany traumatic injury. Nutritional practice in burn injury requires a multifaceted approach aimed at providing metabolic support, while accommodating surgical and medical needs of the patients.

Metabolism after burn injury is mediated through hypothalamic and pituitary axis. There are three phases:

- Ebb Phase/Shock Phase
- Flow Phase/Recovery Phase
- Anabolic Phase

Nutritional care is adjusted to individual needs and is given in three stages.

EBB or Shock Phase

This is usually brief and lasts up to 12-24 hours and occurs immediately post burn. Blood pressure, cardiac output, body temperature and oxygen consumption are all reduced. Loss of skin cover in burn and increased capillary permeability leads to loss of water and electrolytes, mainly sodium and also proteins. As a result, body water shifts from extracellular spaces in other parts of the body to the burn site adding to the continuous loss of fluids and electrolytes. Capillary fluid shift leads to hypotension, hemoconcentration and reduced urine output. Intracellular water is also drawn out to balance extracellular fluid losses leading to cellular dehydration. Patients with extensive burns need immediate fluid and electrolyte replacement during first 12-24 hours. A balanced salt solution such as Ringer lactate solution is given to correct hypovolemia and prevent metabolic acidosis.

A colloidal solution such as albumin or plasma is not effective at this stage as it passes into the extravascular fluids due to the increased capillary permeability which returns to normal only after the first day. During this initial period, fulfilling the nutritional requirements of protein and energy are not attempted as the entire focus is on rapid and effective fluid and electrolyte therapy so as to prevent shock. The goal is to maintain an adequate blood pressure, hematocrit and urine output.

Flow Phase/Recovery Phase

Following the restoration of circulating blood volume, patient enters the catabolic or flow phase after 48-72 hours, fluid and electrolytes are reabsorbed into the general circulation and excess fluid is excreted (Fig. 32.1). From 5th to 12th day post burn, metabolism increases greatly and is characterized by abnormal substrate utilization, increased heat elimination and weight loss. The magnitude of post burn hypermetabolism is proportional to the extent of the injury. Metabolic rate may be accentuated by intervening infection. Cardiac output and oxygen consumption is increased with significant increase in blood flow to the burn wound, which is necessary to provide adequate glucose substrate as an energy source for wound healing. The wound preferentially utilizes anerobic glycolytic pathways which are inefficient for energy production.

Large open wounds, increased blood flow to the burn wound and loss of cutaneous vasomotor reflexes for heat retention account for major heat loss via evaporation. This also leads to increased oxygen consumption. To prevent lethal cooling, the core temperature of burn patient is elevated. Wound evaporative losses are estimated by the following formula:

$$\text{Body Surface Area} \times (25 + \% \text{ of burn}) = \text{cubic cm of water loss/hour}^{1}$$

Being an energy consuming process, evaporation adds to energy loss. For example, a patient losing 86 ml of water/hour by evaporation needs 1200 kcal/day in addition to his/her energy requirement. The increase in basal metabolic rate (BMR) is brought about by increased secretion of catecholamines which stimulate the production of glucagon which promotes glycogenolysis and gluconeogenesis and suppresses the insulin release. Protein catabolism (proteolysis) occurs mainly in the skeletal muscles. Alamine and glutamine released from the muscles are transported to liver which disintegrates them into pyruvates and then in turn into glucose (gluconeogenesis). Amino acids are wasted as gluconeogenesis is exceeding the demand of cells. Enhanced lipolysis to increased demand combined with impaired fat oxidation results in futile recycling of free fatty acids and triglycerides. There may be increased excretion of nitrogen (20-25% of total nitrogen loss is from burn wound exudates), potassium, phosphate, creatinine, magnesium and zinc sulfate in urine. In addition, endogenic anabolic activity decreases because of burns. Therefore, if a patient is not getting proper nutrition, the visceral proteins are wasted, the muscle volume declines and, as a result, the body is incapable of adapting to the existing situation and unable to economize the use of energy. With the onset of wound healing, the metabolic rate and the production of catecholamines return to normal levels and protein synthesis follows as the insulin glucagon molar ratio reverses after the wound is healed.

Anabolic Phase

During this period, the patient is well hydrated and the reactions due to metabolic stress are under control. The patient may still be hypermetabolic and has depleted reserves of all nutrients. Rigorous nutritional support during this period is essential to promote fast recovery and proper rehabilitation. Proper nutritional care during this period can help in preparing the patient physically for undergoing successful skin grafting.

Rate of utilization of nitrogen does not differ in flow or anabolic phase but the obligatory nitrogen loss and the requirements for nitrogen equilibrium decrease as the post burn course progresses through convalescence.

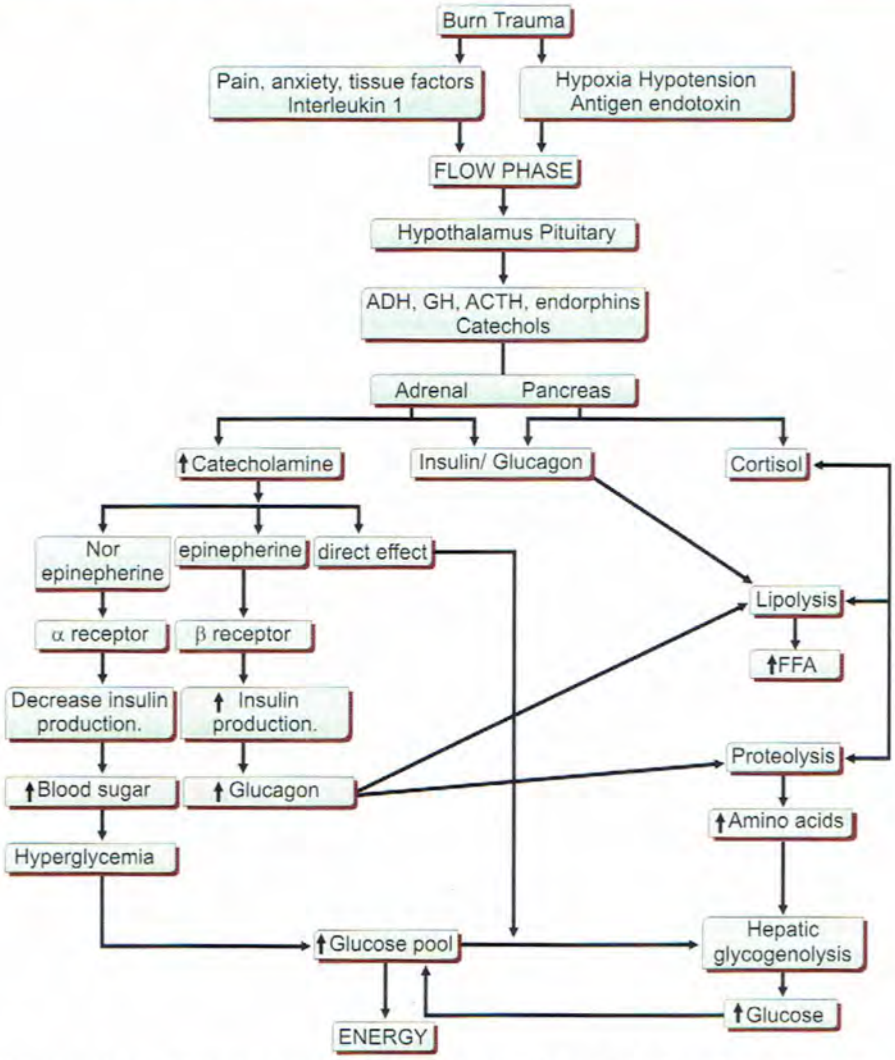


Fig. 32.1: Hormonal changes following burns (*Adapted and modified from:* Total Burn Care by David Herndon, 1996 and Hand Book of Burns Management by Ramakrishnan and Hanumadas, 1991)

PARAMETERS TO ASSESS NUTRITIONAL STATUS OF BURN PATIENT

In the acute phase of injury, driven by series of inflammatory mediators, catecholamines and counter regulatory hormones, the nutritional status deteriorates regardless of initial baseline. Though the body weight which is a reasonable marker of fat and lean tissue is the most useful assessment tool for nutritional status, it has limitations as post burn weight is usually increased due to resuscitative fluid administration, dressings and edema, etc.

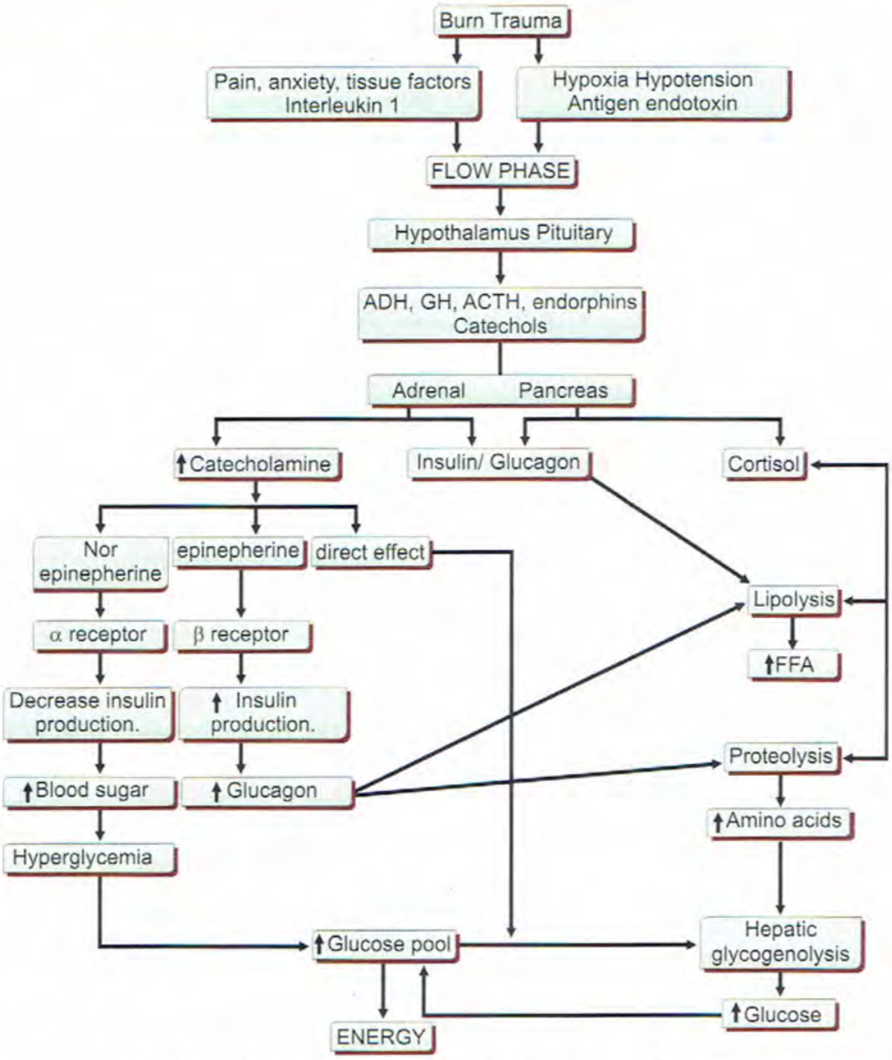
Upper arm anthropometry (upper arm circumference, triceps skin fold thickness) is not a valuable assessment tool in massive burns with edema. This is especially so, if burn wound eschar and underlying wound edema involve the areas to be assessed.

Circulating proteins or visceral proteins are good indicators of nutrition.[2,3] Serum albumin less than 3.5 g/dl and prealbumin less than 1.2 g/dl indicates malnutrition. However, they are also affected by various other factors like rate of protein synthesis and breakdown, exudative wound losses and blood transfusion(s).

Some investigations which can be done to assess the nutritional status are:

- Nitrogen balance studies
- Urinary creatinine excretion
- Estimation of total body potassium and nitrogen
- Serum transferrin estimation
- Indirect calorimetry

They have their own limitations. Pre-injury height and weight of patient or daily body weight and clinical



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Some investigations which can be done to assess the nutritional status are:

- Nitrogen balance studies
- Urinary creatinine excretion
- Estimation of total body potassium and nitrogen
- Serum transferrin estimation
- Indirect calorimetry

They have their own limitations. Pre-injury height and weight of patient or daily body weight and clinical appearance is most effective index of nutritional balance. Estimation of hemoglobin, serum proteins, albumin-globulin (A/G) ratio, immunological markers–total lymphocyte count and delayed hypersensitivity are other investigations used to assess the nutritional status.

The pre-burn weight or ideal weight can be estimated by using following equations:

For males – 106 lb for 1st 60" height +6 lb for each additional inch

For females – 100 lb for 1st 60" height + 5 lb for each additional inch

Serial measures of resting energy expenditure by indirect calorimetry, if available, is the best method to assess the nutritional status as energy requirements vary not only from patient to patient but also from one burn unit to another, based on the standards of burn practice.[4]

NUTRIENT REQUIREMENT AND DIETARY MANAGEMENT

Nutritional status is directly connected with prognosis and perspectives of burned patient.

After resuscitation and assessment of nutritional status, the patient is examined for the presence of diabetes, hyperlipidemia, any bowel or liver disease, alcohol abuse and lactose intolerance, etc.

Although early feeding, i.e. within 24-48 hours after an acute event, is preferred, it is better to rely on IV fluids or clear oral liquids for first two days as the narcotics used for pain relief during resuscitation may cause nausea, vomiting and anorexia. Fluid and electrolyte requirements are calculated considering the daily maintenance, burn wound losses, losses due to fever and through gastrointestinal losses (Table 32.1).

Early parenteral nutrition has no place in Intensive Care Unit, in patients without pre-existing malnutrition. Early enteral nutrition is beneficial in patients with major burns.

As soon as the patient is hemodynamically stabile, other liquids and semisolids are introduced, setting tolerable levels of intake that supports adequate wound healing and weight loss.

Protein and Energy Requirements

By the end of the flow phase the patient usually is well hydrated and has body weight close to the pre-injury weight. The return of bowel movements is an indication that the patient can now be slowly introduced to the nutritional

Table 32.1: Nutrition in ICU patients after the patient is out of shock

Main Food Groups	*Weight /Measures*	*Calories*
Clear Liquids		
Water +Glucose(1tsf)	100 ml	60 kcal
• Orange Juice	200 ml	30 kcal
• Tomato Soup	200 ml	87 kcal
• Soft Drink	200 ml	120 kcal
Switch on to other liquids if patient tolerates clear liquids i.e. 2,500 ml /day		
Other liquids		
Milk + 2 tsf sugar +2 tsf protein rich powders (commercially available)	100 ml/hour	2400/24 hours
Lentil Soup	200 ml	137 kcal
Vegetable Soup	200 ml	100 kcal
Semisolids		
Kichadi (Rice with Lentil)	150 gm	190 kcal
Porridge (Dalia)	2 bowls	410 kcal

I/V fluids after 24 hours (maintenance fluid) =50 ml /kg +evaporative water loss
(Evaporative water loss = 4ml/kg × 20 × % burn/100)
Intravenous alimentation till patient is on full diet
Dextrose 10% 1000 ml = 400 kcal
I/V fat preparation 10% 500 ml = 450 kcal
Amino acid preparation 3.5 to 5 % (200 ml) = 1 gm Nitrogen
Total volume = 1900 ml
Non-protein calories = 850 kcal
Calorie-protein ratio = 150/200: 1 gm Nitrogen
6.25 gm protein = 1 gm Nitrogen

support in the form of oral intake, enteral feeds or parenteral nutrition. A combination of any two of these routes is required on a day-to-day basis depending upon the changing clinical parameters and the severity of burns. Dietary management should be such as to fulfill the following objectives:

- Providing enough calories to prevent subsequent weight loss.
- Maintaining fluid and electrolyte balance.
- Minimizing stress response to pain and anxiety
- Maintaining a positive nitrogen balance
- Replenishing the depleted reserves of vitamins and minerals
- Preventing Curling's ulcers, ileus and other complications

Energy

The energy needs of the burn patient varies according to the depth and size of the burn. According to Robert et al, of all the major forms of trauma, it is the burn injury that causes the maximum disorder of metabolism.[5] The requirement is highest in 3rd° burn. Several formulae have been developed to determine the energy needs. Long's modification of HARRY BENEDICT Formula may be used for calculation of energy needs. However, the results obtained are not definite as the burned area, its depth, localization and possible presence of infection are not included in the formula. Curreri formula is most commonly utilized and is mentioned below (Tables 32.2 and 32.3). Calorie requirements should be estimated based on wound size and patient weight.[1]

Calories needed per day = 24 kcal × Kg (wt) + 40 kcal × % total body surface area (TBSA) burn (Using a maximum of 50% burn)

The demand of energy in patients with burns may be very high. It is important to keep a rational balance and not overload the patient with huge amounts of energy, which will not be assimilated but accumulated as fatty layer beneath the skin. As per a study published in 2002 by DW Hart, if the amount of energy provided is in excess, it leads to greater complications and increased mortality.[6]

SCHOFIELD Equation can be used in which calories are adjusted according to the stress level.

Energy = 25 to 30 kcal/kg body weight/day + adjustment according to stress level

In Burns Energy = 25 kcal/kg/day + 20 kcal/% burn.

Contribution Towards the Total Energy through Various Macronutrients/Micronutrients

Carbohydrates

To fulfill calorie requirements, the first priority is given to carbohydrates which should provide 60-65% of total energy. Adequate carbohydrate supply reduces protein requirement for gluconeogenesis and protein is spared for wound healing and to enhance the immune status. Rate of administration of glucose should not be above 7 mg/kg/min, as glucose above this rate will not be utilized to release energy but will be converted to fat. Blood glucose levels should be closely monitored to prevent hyperglycemia and its associated complications.

Table 32.2: Curreri formula I for open wounds (size less than 20 % BSA) (*Adapted from* Handbook of Burn Management, Ramakrishnan and Hanumadas, 1991)

Age	*For Open Wounded (Size Less Than 20% BSA)*
1-3 Years	(110 × kg body weight) + (40 × % burn) = Kcal/Day
4-6 Years	(85 × kg body weight) + (40 × % burn) = Kcal/Day
7-10 Years	(80 × kg body weight) + (40 × % burn) = Kcal/Day
11-14 Years	(55 × kg body weight) + (40 × % burn) = Kcal/Day
15 Years Onwards	(35 × kg body weight) + (40 × % burn) = Kcal/Day

Table 32.3: Curreri formula II for open wounds (size more then 20% BSA) (*Adapted from:* Handbook of Burn management, Ramakrishnan and Hanumadas, 1991)

Age	*For Open Wounds— Size More Than 20% BSA*
1-3 Years	(100 × kg body weight) + (40 × %) = Kcal/Day
4-6 Years	(70 × kg body weight) + (40 × %) = Kcal/Day
7-10 Years	(60 × kg body weight) + (40 × %) = Kcal/Day
11-14 Years	(50 × kg body weight) + (40 × %) = Kcal/Day
15 Years Onwards	(25 × kg body weight) + (40 × %) = Kcal/Day

During the anabolic phase when the patient can eat orally and has normal bowel movement a combination of simple and complex carbohydrates may be given. Providing good amounts of foods rich in mono and disaccharides as well as starches help in preparing meals which are nutrient dense, have small volume and are easy to digest. Thus, rice, refined wheat flour, semolina, sago, arrow root rice, rice flakes, rice puffs, pasta, dextrose, glucose, honey, potato should be incorporated liberally in the diet.

Carbohydrates provide 4.1kcal/gm. The number of non-protein calories given should bear a definite relationship to the nitrogen intake. The mix should provide approximately 150 kcal/gm of nitrogen or a calorie to protein ratio of 20:1 (normal equals approx 300 cal/gm of nitrogen).

Fats

15-20% of fats is an excellent source of calories (9 cal/gm) with polyunsaturated fats providing 4% of the total calories and avoiding a fatty acid deficiency. There is ample evidence to show that the total number of calories is not the critical issue, but the composition of the mixture is. For instance, both carbohydrates and insulin or amino acid administration have the desirable protein sparing effect. Administration of lipids should be carried out carefully in all critically ill patients. Attempting to give all calories by peripheral IV fat preparations does not spare protein. Not more than 20% of non-protein calories in the catabolic phase should be fat. Increasing fat and decreasing carbohydrate percentage in the diet is particularly helpful in trying to wean a patient from the ventilator as carbon dioxide production is decreased. A careful monitoring of immune function, feeding tolerance and serum triglycerides is required during lipid administration as it produces thrombogenic and immunosuppressive effects. Structured lipids and medium chain triglycerides are currently being preferred. Because of impaired gastrointestinal function among many patients it is advisable to lay emphasis on foods rich in emulsified fat and medium chain triglycerides. Nutritional support with lipids helps in wound healing, cellular integrity and absorption of the fat soluble vitamins.

Proteins

It is the most important macronutrient which determines the ultimate outcome of burns. Sufficient quantity and quality of amino acids is required to prevent efflux from the skeletal muscle and maximize protein synthesis for optimal wound healing and immune function.

Fluid loss from a burn wound may be considerable and can contain 4-6 gm protein/100 ml, representing 25-50% of total nitrogen loss. Nitrogen losses via feces have been estimated to be around 1-3 g N/day. Thus 20-25% of total calories should be provided as protein in adult patients.

Protein intakes approaching 1.5 g/kg/day are associated with net balance between synthesis and breakdown.

High biological value intact protein food sources such as eggs, milks, cheese, yoghurts, marine food, meat, poultry, legumes and pulses should be included liberally but in an easy to digest form.

Calculating Nutrient Requirement

NPC–Non-protein calories

Baseline calorie requirement as per Harris- Benedict Equation:

Males-**BEE** =66+ (13.7W) + (5H)-6.8A

Female-**BEE**=55+ (9.6W) +1.8H-4.7A

BEE= Basic energy expenditure

W=Weight in kg, H=height in cm, A=age in years

Shofield Equation= Baseline calorie requirement +/– other adjustments

= [25 +30 kcal/gm/day + other adjustments according to stress level (Table 32.4).

Table 32.4: Protein and energy requirement according to stress level

Stress level	*Proteins in gm/kg/day*	*Energy in kcal/kg/day*
Unstressed	0.8	25
Mild	1.2	25-30
Moderate	1.3-1.75	30-35
Severe	2	35-40
Burns	2.0-2.5	25 kcal /kg/day + 20 kcal/% BSA

Other Adjustments (NPC)

Undernourished	+ 10%
Age 18 to 30 years	+ 10%
Fever	+ 10%/degree C rise
Mobile in bed	+ 5%
Mobile	+ 10 %
Obesity	– 10%
Age > 70 years	– 10%

For optimal utilization of proteins, adequate calorie supplementation is essential.

Vitamins and Minerals (Micronutrients)

Exact requirements of the micronutrients are not known. Supplementation is aimed at correcting a deficiency state. It is generally advisable to give plenty of fresh fruits and vegetables if an oral intake is feasible. Supplements of calcium phosphate, zinc and iron are generally required. Packed red cells may be required to treat anemia. Recommended doses of vitamins and minerals in moderate and severe burn patients are given below.

Vitamin C – 500 mg/day
Vitamin K – 10 mg/week
Iron – 10-15 mg/week
Electrolytes

- Na^+: 1 to 2 mmol/kg/day
- K^+: 0.5 to 1 mmol/kg/day
- Mg^{++}: 0.35 to 0.45 mEq/kg/day
- Ca^{++} : 0.2 to 0.3 mEq/kg/day
- PO_4^{2-} : 20 to 30 mmol/day

(Vitamins: Standard formulae meet broad range of requirements)

Fluid requirement—Baseline water requirements in adults may be taken as 30-35 ml/kg/24 hour or approx 1 ml/kcal/24 hour (25 ml/kg/24 hour for those who are above 65 years).

Additions must be made for fever approx 300-500 ml/kg/24 hour/degree C above normal and other losses. Fluid restriction may be indicated in certain disease states such as cardiac, liver and renal malfunction.

TIME TO START NUTRITION

After 48 hours of fluid resuscitation, depending on the gastrointestinal status, nutritional supplementation should begin. Oral route is preferred, supplemented with enteral or parenteral supplementation. Aim is to prepare the wound for cover and patient for surgical stress. By the end of one week vigorous nutritional support should be started to achieve positive energy and nitrogen balance.

Early versus Late Enteral Nutrition

The time when nutritional support is initiated is also decisive in the prognosis of burn patients. Proponents of early nutritional support say that enteral feeding started within six hours of injury is safe and effective and rapidly reverses several of the most important metabolic and hormonal effects of burns.

It was stated above that enteral feeding of the burned patient should be started only after the period of shock is over, i.e. the second or even the third day after the burn. Recently, an early enteral nutrition has gained more support. It must be started in six hours after burns. In developing countries, it is difficult to treat and dress all the burn patients in isolated wards and assure the medical care in aseptic conditions. While treating severe deep burns, the problem of wound covering and donor areas also arises. Consequently administering the enteral nutrition earlier helps in reversing the most important metabolic and hormonal effects of burn. First studies showing advantages of an early nutrition were conducted in 1994. Gianotti has shown that early enteral nutrition lessens the translocations of bacteria.[7] The study published by Wang (1997) showed that early enteral nutrition lessens catabolism after burn. The need for energy in burned patients was studied, as well as glucagon, serum insulin, urine cortisol and catecholamines. Decrease of these indices has been proven to be statistically reliable in the group with an early enteral nutrition compared to the later enteral nutrition group.[8]

It is recommended to start enteral nutrition with hypocaloric mixture (0.5 kcal/ml) at the rate 25 ml/hour. If nutrition is well tolerated by patient, the rate is increased by 25 ml at every 8 hours, to 100-120 ml per hour.

Once we are aware of the nutrient requirements of an extensively burnt patient, let us take a closer look on the methods we can use to feed our burn patients.

Modes of Feeding/Nutritional Support

Oral Dietary Support

Oral feeding should always be encouraged and attempted, if tolerated by the patient as this is the most convenient and natural way of feeding. Other modes of feeding should be used only if this route is ineffective or cannot be used. Diets should be designed to give the calculated calorie value and protein. Acceptable protein intake can be obtained with selective high protein, high calorie diet and with additional supplements of high protein liquids between meals. Concentrated oral liquids with protein hydrosulfates or amino acids must be given to ensure adequate intake (See Appendices 2.1 to 2.3).

Most of the patients are depressed and may need encouragement by family and medical team to promote adequate food consumption. Food should be attractive and appetizing and individual likes and dislikes must be considered. It should include all the nutritional requirements.

Meals should be served at regular intervals. The importance of adequate nutrition should be explained and stressed to the patient and his/her relatives.

We should start with liquid diet and gradually switch to semi solids and then solid foods in 4 to 5 days. Cleanliness in the preparation and serving is of paramount importance to avoid GIT disorders. Enfeebled patients and patients with burned upper extremities require assistance. Excessive lactose in diet may precipitate post-traumatic lactose intolerance leading to bloating of the abdomen, flatulence, cramps and diarrhea. Burns of oral cavity may preclude feeding by oral route and may require alternate route of nutrition. Moreover, oral feeding solely cannot meet the elevated calorie requirement.

Anabolic steroids such as oxandrolone are often combined with high protein diet to promote weight gain and enhance anabolism in these patients.

Enteral Nutrition by Tube Feeding

Administration with nasogastric tube is recommended as this is most physiological. If large amounts of enteral mixtures need to be administered, nasoduodenal or nasojejunal tubes may be used. For long term enteral nutrition percutaneous gastrostomy may be planned.

Enteral nutrition can be judicially used in patients with good bowel functions to meet the extra calorie and protein requirement for more than 20% body surface area of burn patients. It is also indicated in patients who are unable to eat or unwilling to eat due to reasons like sepsis, endotracheal intubations, following anesthesia, etc. Maximum calories that can be provided through this method are limited to 3000 kcal/day combining the calories from protein and non-protein sources (Table 32.5). Small bore tube which is well tolerated and allows for oral feed is preferably used in our center. Small amount (0.5 -1 ml/kg/hour) of low bulk iso-osmolar formula is started initially and gradually the amount is increased. Most of the tube feeding formulae contain a high protein diet homogenized in a blender with milk and supplemented with some type of commercial protein preparation. Eggs, 1 tsf of honey and cube of butter may be added to it. Small feeds should be prepared and given at frequent intervals till calculated volume for 24 hours is achieved. Feeds exposed to room temperature for more than 8 hours should be discarded. Gastric contents should be aspirated every time the feed is given. Gastric residue after 4 hours should not exceed 150 ml or else the feeding should be postponed.

Enteral nutrition can be administered as bolus feeds or by a constant delivery pump. Ideally enteral nutrition can be started at a rate of 15 ml per hour of full strength formula and increased stepwise by 15 ml per hour every 12 hours to reach up to 100 ml/hour. An antiemetic may be added to decrease gastric residual volume.

Instructions to be followed during Enteral Nutrition

- Feeding tube to be placed by expert
- Check position of feeding tube by X-ray.
- Head to be elevated to 30 degree during feeds.
- Aspirate the tube before every feed or 4 hourly if on continuous drip.
- If aspirate < 150 ml return aspirate and start feeding.
- If > 150 ml reduce feeding rate or increase the time interval.
- Irrigate tube with water after every feed or 4 hourly.
- Intake/Output charts to be maintained.
- Electrolytes, serum proteins, kidney and liver function tests to be done twice a week
- Weigh the patient on alternate days
- Once the iso-osmolar formula is tolerated, hyper-osmolar formula may be tried to attain 1kcal/ml. Continuous drip using a mechanical pump may be used for feeding. The feedings are changed to oral dietary support as soon as the patient can take adequate oral diet.

Oral tube is most preferable choice but may not be possible in uncooperative and unconscious patients. Naso-gastric tube feeding is given to patient with swallowing impairment with intact small bowel.

Table 32.5: Approximate combinations of nutrients in Enteral Feeds[9]

Protein(nitrogen)	*Normal 10 to 16 gm*	*Burns 16 to 20 gm*
Carbohydrate	1000 cal	1200 cal
Fat	1000 cal	1200 cal
Total calories (non-nitrogen)	2000 cal	2400 cal
Total volume	2.5 liters	3 liters

Adverse Effects of tube feeding include vomiting, diarrhea, abdominal distention, dehydration, gastric dilatation and aspiration. Monitoring of fluid balance, osmolality and electrolyte status is required to avert the adverse effects. Rectal examination should be done to exclude fecal impaction which may lead to overflow diarrhea. Adequate nursing care to administer tube feeding without complications is absolutely essential. Prokinetic drugs such as metoclopramide may be used.

Enteral feeding offers many advantages over parenteral feeding. Oral feeds help in maintaining integrity of mucosal cell mass which prevents endotoxins and bacterial translocation to the systemic circulation. It also maintains the secretion of digestive enzymes and trophic hormones particularly gastrin which promotes insulin secretion and anabolism.

Parenteral Nutrition (See Appendix 2.4)

Parenteral feeding is required in the following situations:

- In combination with oral/enteral feeds and extensive burns more than 40% TBSA or deep burns more than 20% BSA to meet high calorie needs.
- Gastrointestinal dysfunction or paralytic ileus.
- Patients with septic complications or inhalation injury.
- Patients on ventilatory support.
- Patients undergoing aggressive fluid resuscitation who are at risk for poor intestinal perfusion. Parenteral nutrition can be given by
  1. Peripheral vein
  2. Central vein

Parenteral support via a peripheral vein is considered while increasing the oral/enteral support to the desired goal. For parenteral nutrition, a central vein is preferred over the peripheral veins because of non-availability of peripheral veins in chronic burn patients and early development of thrombophlebitis in peripheral veins following administration of hyperosmolar solution.

The advantages of peripheral vein cannulation are:

- Convenient insertion and maintenance
- Lower incidence of mechanical and infectious complications
- Short term parenteral nutrition of less than 7 days
- Partial (supplementary) parenteral nutrition
- Temporary in the absence of technical expertise

Choose a large vein away from injury.

Advantages of parenteral feeding via central vein are:

- When calorie intake via other routes is insignificant or nil or
- When hyperosmolar liquids have to be given to meet the calorie requirement in extensive burn patients, it is easier to give via central vein as it does not cause thrombophlebitis.

The ideal vein for central venous access is Subclavian vein; internal jugular vein is second best choice. Femoral vein is best avoided though still used frequently.

One cannula or one lumen of cannula should be dedicated for parenteral nutrition only. No other medication should be given through it; blood should not be drawn from this (except for the purpose of culture)

Disadvantages of central vein catheterization are:

- Insertion and maintenance needs expertise
- Higher incidence of infectious and mechanical complications

Central veins are therefore used in:

- Prolonged parenteral nutrition of more than 7 days
- Total Parenteral Nutrition
- Peripheral vein not accessible.

Investigations to be done before starting total parenteral nutrition (TPN)

Hematological

- Blood sugar
- Blood urea/serum creatinine
- Serum Albumin
- Magnesium, Calcium, Phosphorus, Lipid Profile
- Prothrombin time
- Liver function tests(Bilirubin and Enzymes)
- Urine analysis
- Serum electrolytes (Na^+, K^+, Cl^-, HCO_3^-)
- Coagulation Profile

Optimization of fluid and electrolyte status is mandatory before starting TPN. Daily monitoring of blood chemistry is required along with body weight and fluid balance.

TPN can be given as separate components of commercially available preparations of protein, fats and carbohydrate or as premixed multicomponent solutions which are now available and have reduced the potential for error and metabolic aberration see Appendix 2.5 (Tables 2.5A and B).

Maximum rate of dextrose administration should be 4-6 mg/kg/min to avoid the risk of hyperglycemia.

If carbohydrates are given as the only source of energy, they can lead to:

- Deficiency of essential fatty acids
- Deficiency of fat soluble vitamins
- Excess CO_2 load
- Hepatic steatosis
- Increased osmolarity of solution.

Instruction charts should be maintained stating the following details of parenteral nutrition.

- Total energy calculated on body weight basis.
- Substrate distribution (volume of carbohydrate, fat and protein)

- Fluid volume.
- Rate of delivery.
- Hang time.
- Total time frame.
- Steps to be taken in case of accidental change in rate of delivery.

It is said that to prevent the complications of parenteral feeding, like over feeding of intravenous nutrients which may leads to hepatic disorders, fluid retention and metabolic derangements, the required volume for the parenteral nutrition is determined by the rate of substrate utilization as opposed to a predetermined energy goal, as demand of energy may reach to 50 Kcal/kg/day. It is important to keep a rational balance and not overload the patient with huge amounts of energy.

The maximum oxidation of glucose in liver is when given at a rate of 4-6 mg/kg/min. If given beyond this rate, it is not assimilated but deposited as fat beneath the skin.[6]

Proteins at 2.5-4 gm/kg can be given, increasing gradually to the desired goal.

20% Fat preparations are not given during acute phase of shock. Starting with 0.5 gms/kg/hour, the maximum rate of 1.0 to 1.5 gm/kg/day should not be exceeded when given during the recovery phase.

The daily volume for TPN requirement is calculated on the basis of;

- All on-going losses, including insensible loss
- Input–output chart
- Alternatively , 30 to 40 ml/kg body weight
- Caution in patients with renal dysfunction and congestive heart failure

For infants and children < 20 kg =1.7 ml/kg/hour
For children> 20kg =1.5 ml/kg/hour.

This is started at 60 ml/hour and increased to 80 to 100 ml/hour after 4 to 6 hours. PN should ideally be given during the day time. Lipid emulsions when given alone should be completely infused within 12 hours of hanging. Lipids when administered as total nutrient admixture can be hung for 24 hours.

If parenteral nutrition is interrupted due to any reason, clear instructions should be stated to administer 10% dextrose solution at the same rate to prevent sudden hypoglycemia caused by high endogenous insulin. Complications of intravenous hyperalimentation are greater than with enteral route.

Complications of Parenteral Nutrition

Mechanical –Related to catheter placement or thrombosis

Infectious–Bloodstream or catheter site infections

Metabolic

- Hyperglycemia
- Hypertriglyceridemia
- Fluid, electrolytes and acid base disturbances
- Asymptomatic rise of serum hepatic transaminases
- Re-feeding syndrome.

Re-feeding Syndrome

It is a rare life-threatening syndrome, seen when carbohydrate-rich feeding is started in severely malnourished patients. It is characterized by massive influx of electrolytes inside the cells resulting in hypokalemia, hypophosphatemia and hypomagnesemia. The precautions to be taken in such patients are providing only half the calorie needs of one day, administration of thiamine (250 mg in 100 ml 5% dextrose IV over 30 minutes) before starting PN and aggressive electrolyte monitoring.

Warnings and Precautions for PN Use

Patients on parenteral nutrition may develop severe egg allergy or allergy due to high levels of triglycerides (> 400 mg/dl). Rapid infusion of lipid emulsions can cause fever, chills, tachypnea, wheezing, palpitations and vomiting, etc. Discontinuation of PN is required in such conditions of severe allergy.

Weaning of Parenteral Nutrition

- Stop TPN when enteral feeding can restart
- Wean slowly to avoid hypoglycemia
- Monitor hypoglycemia during weaning
- Give IV dextrose 10% solution at 100 ml/hour rate for at least 4 to 6 hours
- Alternatively, wean TPN while introducing enteral feeding and stop when enteral intake meets total energy requirement
- In case PN is to be stopped for some other reason (e.g. a complication), the same protocol of dextrose infusion (10% dextrose @ 100 ml/hour × 4-5 hours) is followed.

Biological Value (BV) related to how much of the protein consumed is actually absorbed, retained and used in the body. More specifically, biological value is the measure used to rate protein quality through nitrogen retention in human. Higher the quality of protein more is the nitrogen absorbed and retained by the body.

Nitrogen Balance: Of the three energy nutrients, carbohydrates, fats and proteins only proteins contain nitrogen. Everyday we excrete nitrogen in our sweat, urine, feces and from open wounds when present. Every hour of the day we are in a state of nitrogen flux. Our body is either in positive, negative or zero nitrogen balance. Zero nitrogen balance is an equilibrium state where the nitrogen we ingest from protein equals the nitrogen we loose. Just before we go into zero nitrogen balance, we must have been in a positive nitrogen balance. This is where nitrogen absorbed and retained exceeds nitrogen used and lost. Only during positive nitrogen balance can our body repair and rebuild muscle tissue, effectively fight infections, recover from illness, handle stress and thereby stay healthy.

In a burn patient, there is an excess of loss of nitrogen from the burn wound and if this excessive demand is not met with, the patient goes into negative nitrogen balance, i.e. catabolic state.

Free Amino Acids versus Intact Proteins

Free forms of amino acids actually compete with one another for their uptake through the intestinal lining. This competition limits the rate of absorption because ingested free form of amino acids compete with each other for absorption; they can overwhelm available transport mechanism, therefore, reducing total amino acid absorption. These amino acids result in the poorest nitrogen retention, and are hence, the least anabolic. Some of these free amino acids have another drawback. They increase intestinal osmotic pressure, which can interfere with the absorption of all the nutrients and lead to diarrhea and intestinal distress.

Proteins that are partially hydrolyzed to form peptides are the most anabolic form of dietary supplements and are absorbed faster than either single amino acids or whole proteins. They create the highest nitrogen retention and do not cause intestinal problems.

ASSESSMENT OF NUTRITIONAL SUPPORT

It is very important to evaluate the efficiency of given nourishment because insufficient nutrition impairs wound healing. Evaluation of nutritional state of the burned patient should be done by clinical analyses. First sign of insufficient nutrition is fatigue of patient while performing everyday procedures, as well as during physiotherapy. Tolerance to enteral nutrition should be constantly observed and in case of tube feeding remaining volume of food in stomach should be measured and intestinal transit should be observed.

1. Intake records gives idea of daily protein and calorie intake.
2. Daily weight record is a simple method of assessing effectiveness of nutritional support. However, it may be inaccurate due to bulky dressings, fluid shifts, excision and amputations. Sometimes, it is difficult to weigh a sick patient.
3. Immunological markers may be unreliable for assessing nutritional status.
4. Serum albumin levels or transferrin can be easily assessed.
5. Nitrogen balance studies –consistently elevated blood urea nitrogen (BUN) may indicate deterioration in renal function or increased protein catabolism. Very high BUN is an indication to reduce protein in the diet. If there is no insufficiency of renal function, nitrogen balance is a good indicator of nutritional state and efficiency. Nitrogen balance is a difference between obtained and lost nitrogen (N). In practice, excreted nitrogen is measured only in urine but it is necessary to specify nitrogen loss while including other physiological ways of excreting nitrogen e.g. excretion from hair and through skin in exudation.

Nitrogen loss = N lost with urine + 8 mg/kg body + 0.2 gm N/% body surface burned.

N urine = (urea in urine x 0.08)/2.14 + 4 gm.

Here 0.08 is a coefficient to convert urea from gm/L into mmol/l, 2.14 is a coefficient of recalculations used to find out the level of nitrogen in urine from urea level in urine.

Nitrogen Balance NB = NI (nitrogen intake) – nitrogen lost in (U+F+S) (urine, feces and skin)

On the first day, blood sugar is measured every 6 hours for 24 hours. During the first week, serum electrolytes, blood urea, sugar and triglycerides are measured daily. Unstable patients may require their blood sugar and serum electrolytes measurements as often as twice a day. Serum calcium, bilirubin, alkaline phosphatase, phosphorus, magnesium and blood counts should be done at least twice a week.

Liver profile and enzymes estimation of serum, trace elements and serum transferrin are done once a week.

NUTRITIONAL ASSESSMENT CHART

See Appendix 2.6 for Stepwise Calculation of Nutritional Requirement.

Surgeons and Residents involved in management of Burns should learn to prescribe a nutritional regimen in a methodic and scientific manner as follows:

1. Before starting nutritional support, patients with shock should be adequately resuscitated and gross fluid and electrolyte imbalance corrected. Mild degree of fluid and electrolyte imbalance can be corrected with ongoing nutritional support.
2. Decide on the route of administration for nutrition, plan the oral/enteral/or parenteral nutrition or partial parenteral nutrition.
3. Calculate the energy requirements of burn patients according to Curreri formula depending on the size of burn wound and age of the patient.
4. These formulae or equations for calorie estimation often overestimate the calorie requirements.
5. To avoid this and simplify it, one can easily calculate the calorie protein needs according to appropriate stress level taking the severity of disease into consideration.
6. Calories are derived from three major substrates – carbohydrates, fats, and proteins. By and large 50% of calories are derived from carbohydrates, 30% from fats and 20% from proteins. In critically ill patients, certain adjustments are required as mentioned earlier.
7. After finalizing the total calorie and substrate requirements, the total permissible liquid intake has to be calculated. Normal recommendation is 30-40 ml/kg/day. However, liquid intake has to be adjusted in specific conditions such as renal and cardiac failure, where volume load needs to be restricted. Consequently, total calories will have to be given in restricted volume of fluid. In patients on total parenteral nutrition, this becomes a challenge and one will have to resort to using more lipid emulsions as they give higher calories on weight to weight basis.

NUTRITION IN SPECIAL CONDITIONS

Pulmonary Conditions

Increased administration of glucose in burn patients > 4 to 6 mg/kg/hour results in increased CO_2 production. Hence, a patient with ventilatory impairment receiving high carbohydrate diet will have harmful effect on pH and respiration. Instead such patients are benefitted by low carbohydrate and high fat diet. Fat should provide 30 to 50% of non-protein calories.

Renal Conditions

Due to massive catabolism in burn patients there is increase in uric acid, potassium, phosphorus nitrogenous wastes. Blood urea nitrogen may increase and complicate the metabolic and nutritional management. Blood urea nitrogen should be kept below 100 mg/dl with protein restricted diets and dialysis. Protein restricted diets containing only essential amino acids may be tried. 0. 6 to1gm/kg/day protein is given initially and gradually increased each day to achieve the tolerated goal. Monitoring the daily increase of BUN levels, if BUN levels increases more than 30 ml/dl/day, amino acids/proteins supply must be reduced and one may consider modified amino acid compositions.

Due to oliguria and hyperkalemia, fluid and potassium restriction is advised in renal failure. Citrus fruits and IV fluids with potassium e.g. Ringer lactate is restricted. If IV fluids are required 5% dextrose saline is preferred. Fluid requirement is calculated on the basis of loss due to burns plus previous day's output. Phosphorus and potassium binders may be used orally.

Because of fluid restriction, optimum non-protein calories are provided with concentrated glucose and lipid solutions. There is reduced glucose tolerance and increased insulin resistance in presence of acute uremia and acidosis hence blood glucose levels should be monitored closely in patients with acute renal failure (ARF). Although there are benefits of using lipids as source of energy in patients with ARF, the intake should be limited to 25 to 30% of total energy.

Hepatic Conditions

In patients with chronic liver disease, aromatic amino acids may lead to hepatic encephalopathy. Supplementation with branched chain amino acids is beneficial in such patients. Milk (casein) and vegetable protein diet should be preferred to meat protein diet with rate of 0.6 gm to 1 gm/kg/day.

However, patients with alcoholic liver disease may benefit from early nutritional support by providing adequate calories, proteins, minerals and vitamins. In the presence of hepatic encephalopathy the total protein intake should be reduced to 0.5 gm/kg/day or even less.

ADJUVANT THERAPY TO MODULATE ENERGY REQUIREMENT AND PROMOTE WOUND HEALING

Environmental and clinical factors that influence metabolism and hence energy requirement

The magnitudes of inflammatory responses are proportional to the severity of trauma and are not preventable.

However, various clinical interventions can affect or amplify this metabolic rate further influencing energy requirements. Due to the combined effects of inflammation and evaporation, the burn patients are amongst the most hypermetabolic. Occlusive dressings, early excision and grafting help in minimizing this effect.

Environmental measures to minimize heat loss by warming the external environment, pain management, sedation, ventilatory support and nutritional therapy contribute to patient's overall energy expenditure. For example a patient, who has good pain control, is well sedated and working little towards the effort of breathing while mechanically ventilated may have lower requirements than a patient who is less critically ill, breathing on his/her own with less sedation and with more participation in rehabilitation. Shizal and Forse documented that carbohydrate was more efficient in diminishing weight loss and protein degradation after injury.

1. Hormonal Therapy

Studies have been done on recombinant Human growth hormone which is a potent anabolic agent.[10] Mortality and morbidity in burns is directly proportional to severity of protein catabolism leading to delay in wound healing, sepsis and multiorgan dysfunction.[11] The hypermetabolic state is related to increase in catabolic hormones and decrease in anabolic hormone.[12-14]

Recombinant growth hormone (rhGH) supplementation in the doses of 0.5 IU/kg/day for 2 weeks together with standard conservative management of burns have shown to arrest severe catabolic state of burns and hence avoid weight loss. Increase in serum albumin and hemoglobin with rhGH treatment further support the anabolic effect of rhGH. The administration of growth hormone also results in marked augmentation of nitrogen retention, decreased BUN and serum basal insulin almost doubles. There exists a close relationship between nitrogen balance and basal insulin secretion and increased insulin augmentation appears to be the controlling mechanism for the improved nitrogen retention.

2. Immunotherapy

Use of Immunoglobulins (particularly enriched with IgM) in burns is now being studied extensively.

It was earlier believed that the task of the immune system was restricted solely to recognition and elimination of viruses, bacteria and their toxins. Today, it is regarded as an organ system which is complex, self regulating and in communication with other systems.

Pentaglobin i.e. IgM enriched intravenous Immunoglobulins have an anti-bacterial, anti-toxic, anti-inflammatory, and immune modulating activity.

The magnitude of inflammatory responses, however, is proportional to the severity of trauma. Adjuvant therapies like giving immunoglobulins can help modulate inflammatory responses, hence affecting metabolic rate which further influences energy requirements. The rate of metabolism increases with onset of wound sepsis leading to an increase in protein and energy requirement. Pentaglobin used concomitantly with antibiotics especially against gram +ve and gram –ve bacteria and their toxins indirectly help in reducing rate of metabolism.

3. Role of Albumin in Burns

Albumin is a naturally occurring plasma protein and has long been thought to be the kind of solution by which patients would most benefit (defined as a "gold standard").[15]

Commercially available human albumin solutions (HA) contain 96% of albumin. Purified protein fraction contains approximately 83% albumin.

The molecular weight of albumin is 69,000 Daltons (approx.). 4% albumin is slightly hypo oncotic, 5% albumin is iso oncotic, whereas 20% and 25% solutions are markedly hyper oncotic, so that total plasma volume is expanded by shifting of fluid from the interstitial/intercellular to the intravascular compartment. 100 ml of 25% albumin increases intravascular volume to a total volume of approximately 450 ml.[16]

The volume effect of albumin is not predictable and depends on blood volume, proteins level and capillary permeability.

The serum albumin concentration is a function of its rates of synthesis and degradation and its distribution between the intravascular and extravascular compartments. The total body albumin pool measures about 3.5 to 5.0 gm/kg body weight (250-350 gm for healthy 70 kg adult). The plasma compartment holds about 42% of this pool, the rest being in extravascular compartment. Some of this is tissue bound and is, therefore, unavailable to the circulation.

Critical illnesses like burns alter the distribution of albumin between the intravascular and extravascular compartments. There are also changes in the rate of synthesis and degradation of the protein. The serum albumin concentration often decreases dramatically early in the course of critical illness. It does not increase again until recovery phase of illness. The kinetics of albumin given IV differ greatly between critically ill patients and healthy subjects. The implication of this fact, given the important functions albumin has in health, is that using exogenous albumin to increase the intravascular albumin concentration during critical illness is beneficial.

Unfortunately, studies have failed to show any benefit of albumin over other colloidal therapies in adult.

The altered distribution in critical illness is related to an increase in capillary leakage. This occurs in sepsis, burns and after major surgical stress. It involves dysfunction of the endothelial barrier, resulting in capillary leakage and loss of protein, inflammatory cells and large volumes of fluid into interstitial space.

The rate of albumin synthesis may be significantly altered in the critically ill. In the acute phase response to trauma, burns, inflammation or sepsis, there is an increase in the gene transcription rate for the positive acute –phase proteins such as C-reactive protein, and decrease in the rate of transcription of albumin mRNA and the synthesis of albumin. IL-6 and TNF-α both act to reduce gene transcription. A sustained inflammatory response in critical illness may lead to prolonged inhibition of albumin synthesis.

Catabolism of albumin may also be altered.

Indications for albumin administration:

1. *Volume replacement therapy:* At present there is no evidence from randomized controlled trials that resuscitation with albumin and plasma protein fraction (PPF) improves survival, compared with resuscitation with cheaper alternatives such as saline, in patients with hypovolemia, burns and hypoalbuminemia, except in highly selected populations of critically ill patients.
2. *Support of colloid oncotic pressure:* It is believed that the oncotic force of concentrated human albumin may help reduce tissue edema. Recent literature states that albumin may exert a direct protective effect in the critically ill patient and its ability to sustain oncotic pressure may only be one of many possible mechanisms.
3. *Maintenance of serum albumin levels:* The normal serum concentration of albumin is 35 to 50 g/l: Albumin appears to be a non specific marker of the seriousness of an illness.
4. As a transport molecule for various drugs and endogenous substances and also acts as an effective plasma buffer.
5. To assist in coagulation: Albumin is involved in the coagulation pathways and has an anticoagulant effect which is probably antithrombotic in nature.
6. Free radical scavenging and maintenance of membrane integrity.
7. Management of fluid shifts
   - As an osmotic agent
   - To redistribute fluid during dialysis
   - To improve oxygenation
8. Miscellaneous - to correct metabolic acidosis
   - Neonatology ICU.
   - Liver disease, ascites, malnutrition
   - Starvation
   - Nephrotic syndrome
9. *Severely burned ICU patients:* These patients are a specific group in whom albumin may have a beneficial role. Though it is reported to be harmful in first 24 hours, its use is justified after 24 hours in profoundly hypoalbuminemia patients as it favors resorption of edema.

In burns covering < 15% of body surface area, it may not be necessary, but its use is justified in > 50% TBSA burns. When using albumin in burn patients, synthetic colloids (e.g. hydroxyethyl starch) have been shown to be effective or even superior in increasing CVP, PCWP, DO_2 and VO_2. Surprisingly one study says that no attempt should be made to normalize serum albumin levels in burns. Albumin plasma levels as low as 15 g/l have been seen to be well tolerated in these patients.

Side Effects of Albumin

There are several reasons why albumin supplementation might make things worse for critically ill patients.

1. Cardiac decompensation may occur after rapid volume replacement with 20% albumin as it leads to four fold increase in volume retention.
2. In patients with capillary leak syndrome, albumin may become detrimental when albumin and water cross the capillary membrane and cause or worsen pulmonary edema, thus compromising tissue oxygenation and finally leading to multiorgan dysfunction.
3. The anti-hemostatic and platelet lowering properties of albumin may increase blood loss in postsurgical or trauma patients.
4. Albumin administration in the resuscitation of hypovolemic shock may impair sodium and water excretion and worsen renal failure.
5. Commercially available preparation of albumin contains aluminium and may lead to toxic concentration of ions.

Despite a growing body of systematic reviews, evidence based medicine analyses and consensus, conferences, the utility and safety profile of albumin is still under dispute.

Human Albumin Replacement Therapy in Burns

1. In cases of extensive and severe burns with an increased hematocrit value, normalization of the circulatory conditions can be approached in a very efficient manner with Human Albumin 20% or 25% Immuno diluted with dextrose, levulose or electrolyte solutions with recommended doses:

Adults: 200 to 400 ml Human Albumin 20% Immuno (40-80 g) diluted 1 in 4, corresponding to 800 to 1600 ml of 5% albumin solution.

OR

200 to 400 ml (50 to 100 gm) of Human Albumin 25% diluted to 1in 5 with isotonic electrolyte or dextrose solutions (corresponding to 1000-2000 ml of 5% of albumin solution)

Children: 4 ml Human Albumin 20% per kg body weight Immuno diluted 1 in 4 corresponding to 16 ml of 5% albumin solution

The total dosage over the 24 hours can be determined in accordance with the formula:

2 ml × body weight (kg) × % of surface burn + 1500 ml

2. After the acute stage has been brought under control considerable protein deficiency, largely of albumin may occur. This hypoalbuminemia can be corrected by administration of the following doses:

Adults: 50 ml Human Albumin 20% Immuno (10 g) twice a day

Children: 1 ml Human Albumin 20% Immuno (0.29 gm) per kg body weight.

Additional loss of protein and diminished albumin synthesis might lead to severe albumin deficiency. In debilitated patients stabilization of the protein balance with Human Albumin 20% Immuno may considerably improve the general condition of the patient.

4. Role of Immunonutrition/Immunomodulators in Burns and Critically ill Patients

The link between malnutrition and impaired immune function has been well documented. The importance of some specific nutrients in burns is proved and these substances have got special attention in recent years. They are called immunomodulators. It is determined that these substances have a positive effect on protein metabolism, immune state and function of intestines. These nutrients known as "immunonutrients" include amino acids arginine and glutamine, Omega-3 fatty acids, nucleotides and antioxidants, vitamins and minerals.

Recently much thought has been given to glutamine as an immune enhancer. Glutamine is an amino acid which can be synthesized by virtually all the tissues and it accounts for more than 60% of body free aminoacid pool. During starvation and particularly during stress the glutamine pool of the body is reduced markedly.

Glutamine has many pharmacological effects. In experimental study by Furst [17,] it was shown that keeping nourishment rich in glutamine in catabolic conditions gives a positive effect on nitrogen balance, protein synthesis, decreases proteolysis in muscles, helps to retain integrity of digestive tract, protects from translocation of bacteria, decreases number of complications as well as mortality. The study of Zhou (1999) determined that supplementing glutamine in early stage after burn in doses of 0.5 to 1 gm/kg decreased the risk of complications and improved wound healing. Arginine is also known as a substance affecting metabolism and immunity.

Cyober[18] recommends administering α-ketoglutarate two times per day, altogether 10 gm (two bolus every 12 hours); it decreases catabolism of muscles and myofibril proteins, saves glutamine in muscle, decreases catabolism in intestine and stimulates protein synthesis in liver after burn.

Omega-3 fatty acids (FA) have anti-inflammatory effect; they inhibit interaction of blood vessels endothelium with leucocytes, inhibit synthesis of interleukin 1 and 6. Ratio of omega-6 FA and general FA level is important because synthesis of prostaglandins or leukotriens predominates depending on the ratio. After burns, mixtures of Immunomodulators can be used, glutamine, arginine, α-ketoglutarate, omega-3 fatty acids, selenium zinc cocktails. They can be used as stable mixtures for enteral nutrition.[19,20]

Current thinking is to avoid Immunonutrition in life threatening septic patients with APACHE scores more than 20, where such radical changes in nutrients may perhaps adversely affect the outcome. Instead use it in less serious patients of sepsis or in early sepsis where it is more likely to be of benefit.

5. Role of Environmental and Clinical Factors

There are lots of environmental and clinical factors that influence metabolism and hence, energy requirements. The initiator of the metabolic response to thermal injury is a large defect. Hence, every effort should be made to promote wound healing and establish a properly functioning surface barrier and interface between patient and surrounding environment.

Adequate nutritional support along with closure of wound is associated with a rebuilding of body mass, gain in weight and the patient's return to an active productive life.

6. Role of Early Physiotherapy

Physical activity leads to deposition and incorporation of amino acids into skeletal protein. With the help of a physiotherapist, a planned exercise programme must be instituted as soon as possible in burn patients. Simple isometric exercises can be accomplished to improve the vitality of muscles and encourage the movement of nitrogen to skeletal muscle cells once the calorie and nitrogen equilibrium is achieved.

SUMMARY

Apart from the medical and surgical needs of a burn patient, the nutritional aspect should also be given equal importance as nutritional care complements the medical and surgical treatment of any burn patient. Nutritional requirement is met not only by carbohydrates, fat and proteins but also by adjuvant hormonal therapy and immunotherapy. Protein catabolism and mobilization of body fat occurs due to hormonal signals that summons these substrates, apparently for wound healing and energy utilization following burn trauma. The signal for substrate utilization or storage is basically an interaction between insulin and catecholamines. Food intake by the patient stimulates insulin production which diminishes the catabolic effects of increased nervous activity and favors protein biosynthesis and glycogen storage. Judicious and early administration of enteral or parenteral feeds in a burn patient goes a long way in reducing the morbidity and mortality in major burns.

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Chapter

33

Physiotherapy for Burn Patients

Sarla Passi

INTRODUCTION

Physiotherapy occupies a very important role in the overall management of burn patients. Irrespective of the extent of burn injury, most minor or very extensive, a burn patient needs physiotherapy. A physiotherapist working in the department of burns has a special understanding of the magnitude of burns, its systemic consequences, the varied complications and the knowledge of the healing processes. He/she can better understand the rehabilitation goals, modalities of treatment and behavior of the patient and ensure quicker healing and rehabilitation.

Post burn deformities in children can be very severe as the bones are still growing and are very soft. The bony deformities can occur due to contraction of the burn scar. The importance of physiotherapy is not understood by most patients and they tend to avoid it because it is time consuming, painful and requires a lot of hard work both by patient and physiotherapist.

The treating burn surgeon must emphasize the need for physiotherapy to the burn patients starting from the initial stages of treatment to ensure minimization of scars, return of complete joint mobility, muscle strength and an outcome of soft and supple scars. Physiotherapy plays a special role in the management of acute burns as well as the post burn sequelae. After any surgical procedure under general anesthesia, the patients require breathing exercises. If the operated area requires immobilization, the rest of the body needs to be exercised. The Physical therapy program involves regular evaluation and supervision followed by thorough planning of the treatment with documented progress report to achieve maximum recovery from the burn injury sustained, in the shortest span of time.

OBJECTIVES OF TREATMENT

The primary goal of physical therapy is to:

1. Make the patient comfortable in proper position,
2. Encourage breathing and coughing to look after pulmonary health,
3. Prevent contractures by splinting and positioning,
4. Maintain normal range of motion of all joints, whether overlying skin is burnt or not,
5. Maintain muscle power,
6. Build positive attitude towards treatment, thus strengthening the confidence of the patient and
7. Modify scars till they become soft and supple.

INITIATING TREATMENT

Physiotherapy starts from the first day. A post burn patient is in considerable pain; hence the first challenge is to address the patient's fear of moving a burnt extremity because of the anticipated pain. When the patient is in a position to sit, he should be taught a comfortable way to sit, fully supported in the beginning and then gradually on his own. If the patient is ambulatory, he should be encouraged to ambulate till he/she becomes independent.

STAGES OF THE TREATMENT

Physiotherapy treatment can be classified into following stages:

a. Acute Treatment, in the *Intensive Care Unit*. This comprises of breathing exercises, proper positioning, supportive movements, splinting and ambulation.
b. Subacute, in the *Hospital Ward*. This comprises of positioning, maximising joint mobility and muscle

strength, ambulation, looking after spontaneously healing and skin grafted areas and advising pressure garments and splints.

c. Follow-up, in the *Outpatient Department*. This comprises of ensuring maximum joint range, maximum muscle strength and return to normal duties, care of skin grafted and spontaneously healed burnt and donor sites.

Physiotherapy in Acute Stage

Day 1

If the neck and chest or back are burnt, the patient may not be able to breathe properly due to pain. The easiest way to encourage the patient to breathe normally is to ask the patient to blow as much as possible. In case of children, blowing balloons may be a good incentive. If a balloon is not available, make do with a paper bag and make it a game to be enjoyed. Deep breathing facilitates the expansion of the chest, which in turn, helps increased oxygen intake (Fig. 33.1).

Before starting any exercises, it is essential to pay attention to the position of the patient. The head end of the bed is raised so that patient is upright and properly supported.

A pillow is placed under the knees to make the patient comfortable, making sure that the diaphragm is not stretched. Ask the patient to blow as much as possible. In case of burned axilla the arms should be raised and supported at 90° from the torso. Standing sideways, the therapist can support the patient's arms.

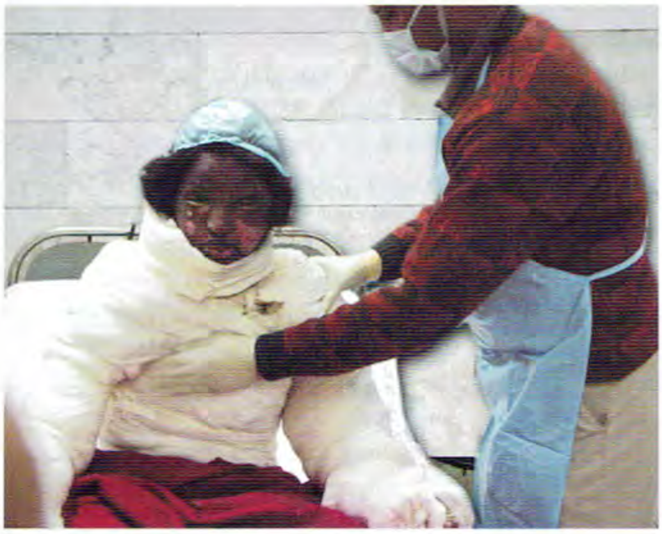


Fig. 33.1: Patient is encouraged to sit and do the breathing exercises

On Day 2

If the patient is not severely burned or if the patient is comfortable and confident, make the patient sit sideways. High sitting (sitting straight with legs down) with the same exercises (as above) can follow (Fig. 33.2).

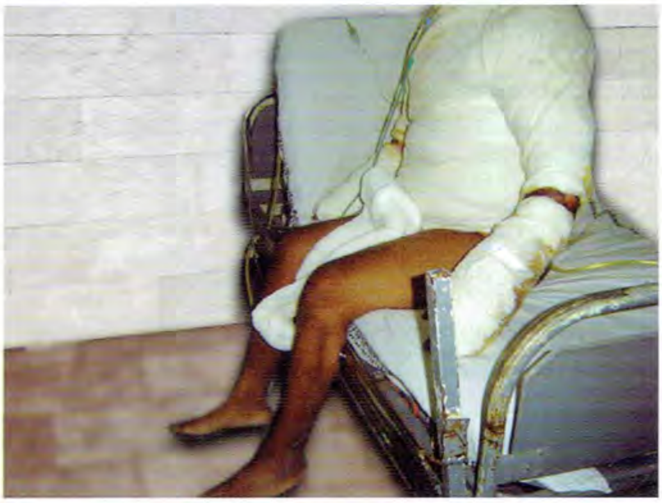


Fig. 33.2: High sitting posture

On Day 3

The same exercises as on *Day 2* continue. The patient can be asked to stand and take a few steps close to the bed. If the patient complains of dizziness (which is normal effect of increased gravitational pull felt on being put in a vertical posture after prolong duration in the horizontal posture during the initial stages of treatment), he should be allowed to lie down immediately. Gradually standing time can be increased.

Day 4 Onwards

The above exercises are increased gradually till patient adjusts to them and with continued exercises the patient will ambulate earlier and feel better. If the patient is severely burned, ambulation may take more time. The exercise program must be continued regularly. Physiotherapist should continue with p*ositioning and splinting in acute stage to prevent contracture later on.*

Management of Neck Burns

Burn injuries in the neck are very common in India, since kitchens in most Indian villages and in poor urban homes have the cooking stoves on the floor. Working on such a stove requires the cook to bend in front of the open flame. In addition, most of these stoves use kerosene to ignite a fire. The resultant burst of flames has a high probability of touching the cook's loose clothing ('dupatta'), usually wrapped around the neck. To make matters worse, these 'dupattas' are usually made of synthetic material, causing even more damage since the material sticks to the neck on catching fire, causing deep neck burns.

It is critical to pay particular attention to the position of the neck, since the neck can flex easily. Although a pillow provides some comfort to the patient, the use of a pillow must be discouraged as it leads to flexion contracture. The patient should also be provided with a cervical collar, after the dressing to prevent flexion of the neck (Figs 33.3 and 33.4).

This may cause the patient to complain of pain in the cervical region, since extensor muscles get fatigued. The Physiotherapist must give static exercises of flexion and lateral rotations to relax the patient and increase strength of muscles (Figs 33.5A and B).

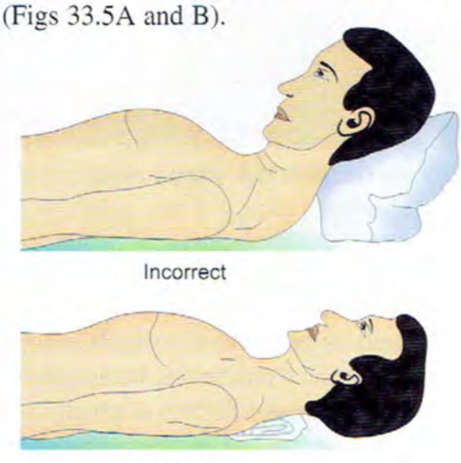


Fig. 33.3: Wrong use of the pillow encourages formation of neck contracture

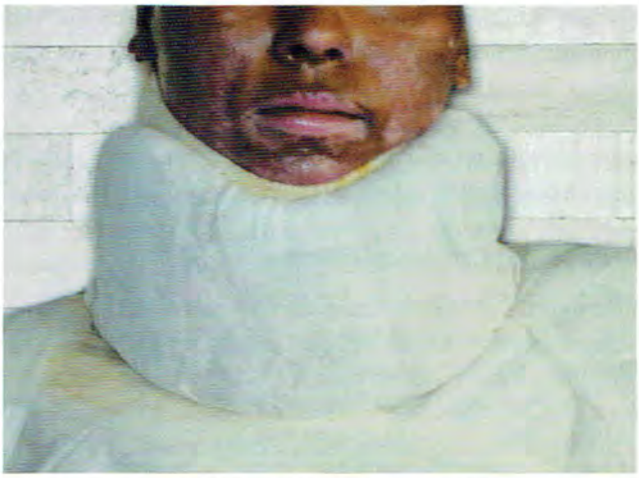


Fig. 33.4: Cervical collar with dressing

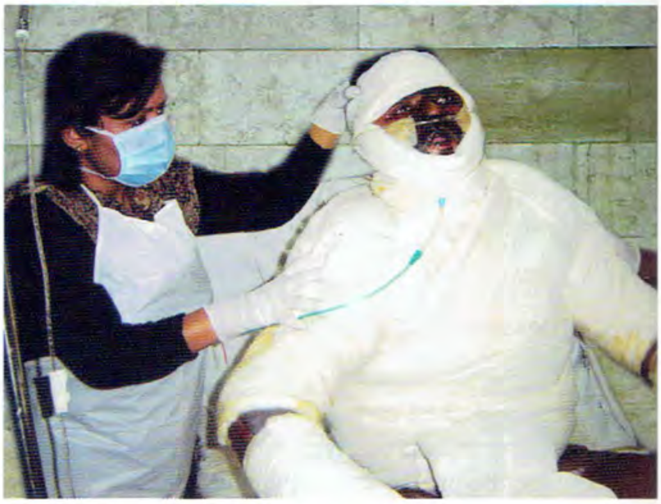


Fig. 33.5A: Neck extension excercises

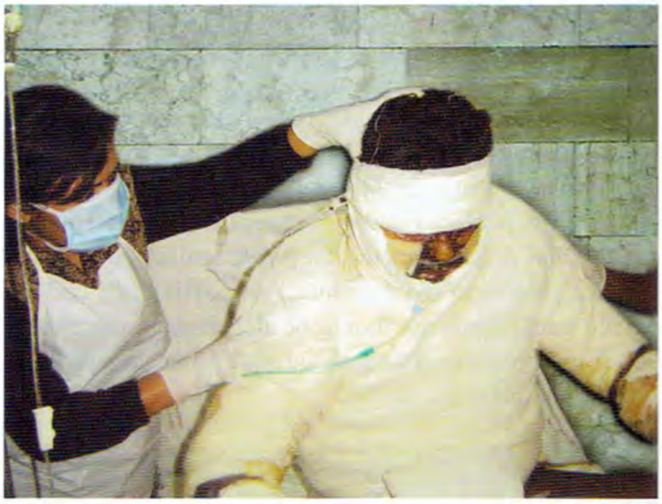


Fig. 33.5B: Neck flexion excercises

Management of Chest and Axilla Burns

A patient with neck, axilla and chest burns needs close attention since the neck and axilla can easily develop flexion contracture. In the case of axillary burns, however, the patient's arm has to be maintained in abduction. A pillow may be used and the patient must be aware of abduction even when he is lying on his side (Fig. 33.6).

In chest burns, breathing exercises play a very crucial role in the complete treatment. The patient tends to avoid breathing properly due to the pain, as a result of which chances of chest congestion go up considerably. Patient should be encouraged to cough to clear the lungs.

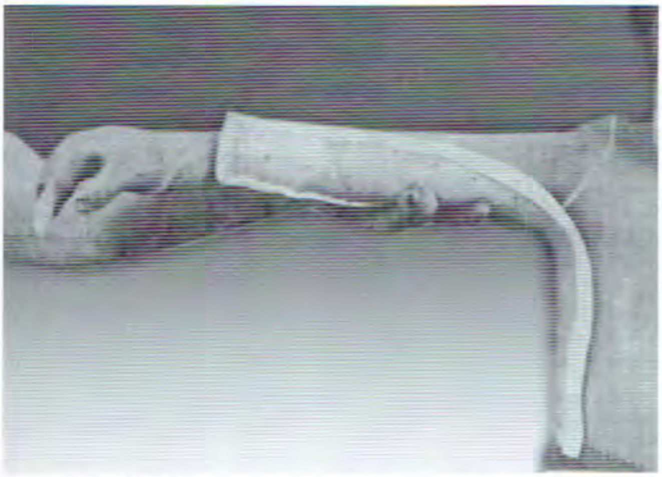


Fig. 33.6: Conforming abduction splint of the shoulder

Management of Elbow and Hand Burns

In the case of elbow burn, the elbow tends to go into flexion contracture very soon. The patient must, therefore, be provided extension splints. In the case of children, it is important to handle the elbow carefully as they may get myositis ossificans. Care should be taken to avoid applying force during dressing and splinting.

The hand is a highly specialized part of the body. Treatment of burns of the hands poses a great challenge to the Plastic Surgeon and the Physiotherapist. The wrist must be kept in extension with Metacarpophalangeal (MP) joints in flexion and Interphalangeal (IP) joints in extension and thumb in opposition (Fig. 33.7A).

After the dressing, a splint must be applied immediately. The splint should be removed twice daily to allow active exercises, after which it is applied again, ensuring that there is sufficient space between the webs and thumb in opposition. Sometimes, ready-made splints may not fit the patient well. In such cases, a customised Plaster of Paris (PoP) splint should be made to ensure that the patient does not develop any contracture (Fig. 33.7B).

Management of Lower Limb Burns

Lower limb contractures are a very common occurrence. If both the hips and abdomen are burnt, the patient is likely to develop hip adduction contracture. This can be very disabling. The patient may not be able to sit, may walk with shuffling gait and may find it difficult to climb stairs. For a patient admitted with such burns, both the legs must be "abducted" and extended, perineal and hip adduction exercises continued. In the case of small children, a hip spica splint may need to be applied. In the lower limb, the splint is given on the posterior side with knee in extension.

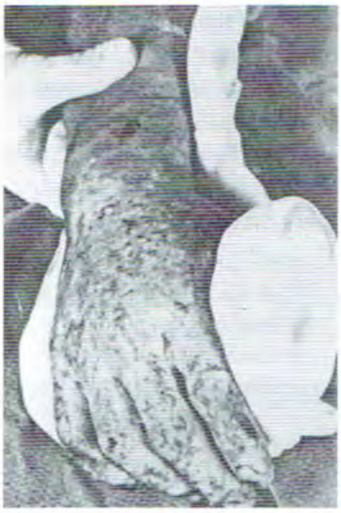


Fig. 33.7A: Position of hand — Wrist in extension, MP flexion and IP extension (Thumb in opposition)

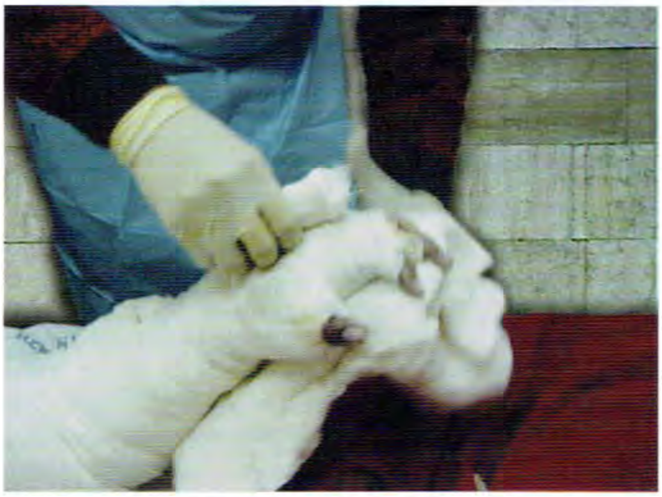


Fig. 33.7B: Simple PoP cock-up splint

Management of Knee Burns

A patient with knee burns must be kept in supine position and given a knee extension splint. After quadriceps exercises twice a day, the splint is firmly tied again. The patient can be ambulated with knee in extension and may be advised to use his hip flexors to clear the ground.

Management of Ankle Burns

Ankle flexion contracture is very common. This is because the inherent strength of the tibialis anterior and pronator tertius muscles enables the patient to start walking on his heels. To stop this, anterior (dorsal) ankle splint should be given. This prevents the contracture and help the patient develop *Heel-Toe* gait. In case of posterior burn, the ankle must be kept in neutral position.

Splinting and exercises must start right from day one or two (Fig. 33.8). If a burn patient has even a few days of immobility in the position of comfort, not only will he/she be fearful and resist any active or passive motion, but musculoskeletal alterations will also begin to develop.

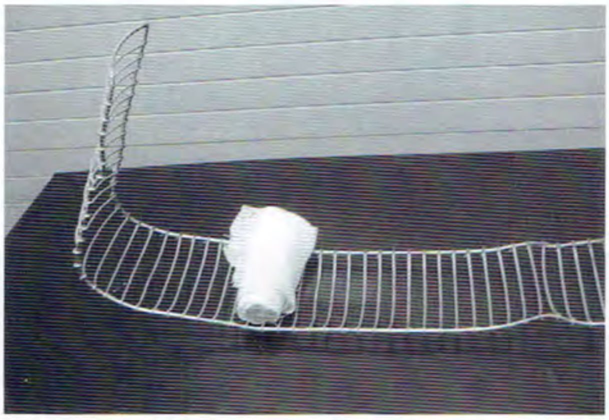


Fig. 33.8: Krammer wire foot drop splint

Physiotherapy in Later Stages

When a patient is under the supervision of a Physiotherapist from the very beginning, there are likely to be fewer complications during the successful treatment. However, in developing countries, a patient suffering from burns may reach a burn center very late. By this time, they already suffer from large raw areas which require skin grafting and/or contracture(s) have already been formed. In such cases, Physiotherapy plays a very crucial role. As soon as patients wounds are completely healed, he/she should be encouraged to eat and dress himself and take care of his personal hygiene. The family should be asked to help him only when the task is impossible. This not only allows physiotherapy of all joints but also gives a feeling of well-being to the patient.

The following steps must be taken to determine the Physiotherapy Program for such a patient:

1. A patient who visits the Physiotherapy Department should be evaluated for the following:
   - How big is the wound?
   - Is it on a joint?
   - Has the patient developed any contracture?
   - If yes, what is the degree of contracture?
   - Has the wound healed with hypertrophic scars?

   If all the wounds have healed by the time the patient reports to the physiotherapist, he has to evaluate all the joints for limitation of motion due to scars and contractures. The severity of limitation is then graded:
   a. *Acceptable:* The motion of the joint is otherwise normal but limited in the final arc of motion (Fig. 33.9).
   b. *Functional:* There is 50% of normal joint motion allowing patient to function independently (Fig. 33.10).
   c. *Severe:* The range of motion is less than 50% of normal, restricting normal function and therefore, demands reconstructive surgery (Figs 33.11A and B).

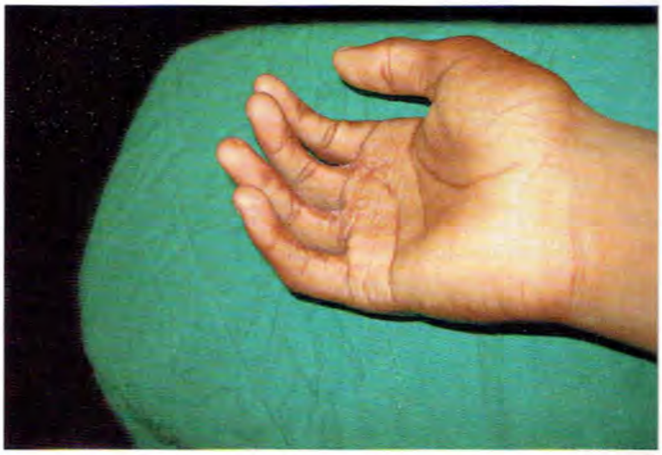


Fig. 33.9: Acceptable deformity

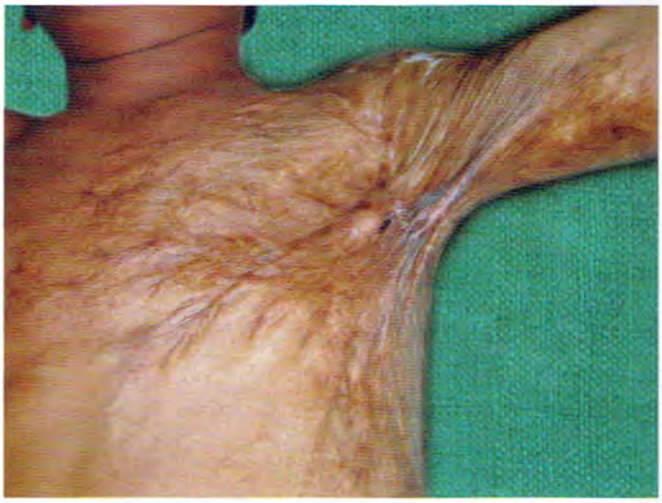


Fig. 33.10: Functional deformity

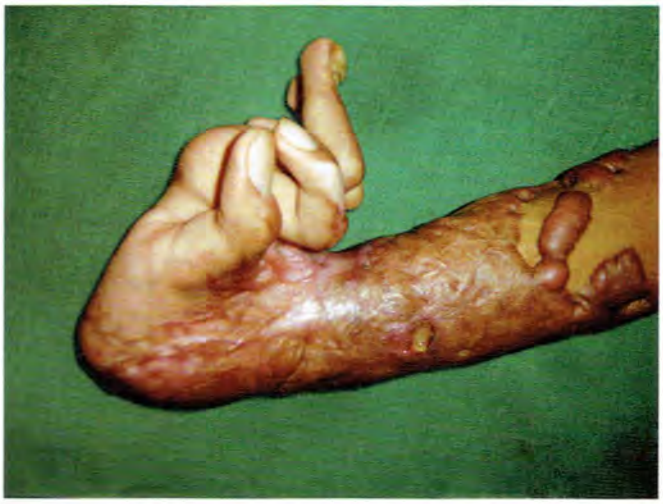


Fig. 33.11A: Severe deformity restricting hand and wrist movements and function

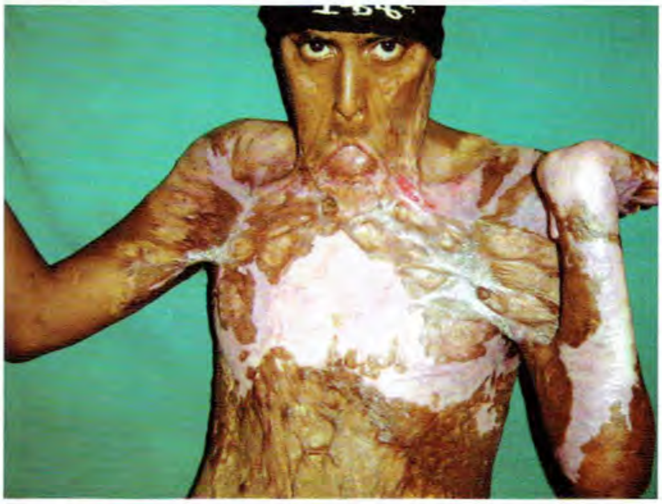


Fig. 33.11B: Severe deformity restricting function of multiple areas

2. After the evaluation, the line of treatment should be determined by the physiotherapist. This includes decisions such as:
   - What should be the exercise program?
   - What splint should be given?
   - How much can the patient achieve?
3. In case of small hypertrophic scars, they may improve with ultrasonic treatment, use of pressure garments and silicone gel.
4. If there is nerve injury, the patient must be evaluated with strength duration curve and reaction of degeneration. A muscle chart is prepared and stimulation given to the patient accordingly. This may be followed by propioceptive neuromuscular facilitation (PNF) exercises.

To understand the role of physiotherapy in the improvement of scars, it is important to recognise the function of skin in the human body:

a. *Regulation of body temperature:* The body temperature is regulated by an increase or decrease in perspiration, superficial blood flow or both;
b. *Protection:* The skin acts as a barrier to protect the body against invasion of micro-organisms, ultraviolet radiation, abrasions, chemicals and dehydration.
c. *Sensation:* Multiple sensory cells within the skin detect pain, temperature and touch.
d. *Excretion:* Sweat and water are excreted and heat is dissipated through the skin.
e. *Immunity:* Normal, periodic loss of epidermal cells removes micro-organisms from the body surface. Immunity cells in the skin transport antigens from outside the body to the antibody cells of the immune system.
f. *Blood reservoir:* Large volumes of blood can be shunted from the skin to the central organs or muscles when needed.
g. *Vitamin D synthesis:* Modified cholesterol molecules are converted to vitamin D when exposed to ultraviolet radiations.

Treatment of Wounds/Scars/Grafts in Physiotherapy

Since the skin covers the musculoskeletal system, any injury to the skin directly affects the movement of that particular part of the body. As a result, the patient tends to keep that part in a comfortable position and not move it in an attempt to avoid pain. Such a comfortable position is very likely to cause contracture and disuse-atrophy. In this stage, as the wound heals, the scars form. The physiotherapist plays a critical role in the improvement of the wound. In the previous chapters, the treatment of wounds with dressing and medication has been discussed in detail. Physiotherapy can help the patient by reducing the pain using ultrasound and ultraviolet therapy that helps in healing of the wounds. Ultrasonic treatment is given around the wound with Pulse duration of 1.5 cm/sec for duration of 5-10 minutes, depending on the area (Fig. 33.12).

Ultraviolet treatment is given by first cleaning the wound with saline. The ultraviolet lamp should be fixed at a distance of 18-20 inches. The lamp should be covered with a cloth so that the other parts of the patient's body are not exposed

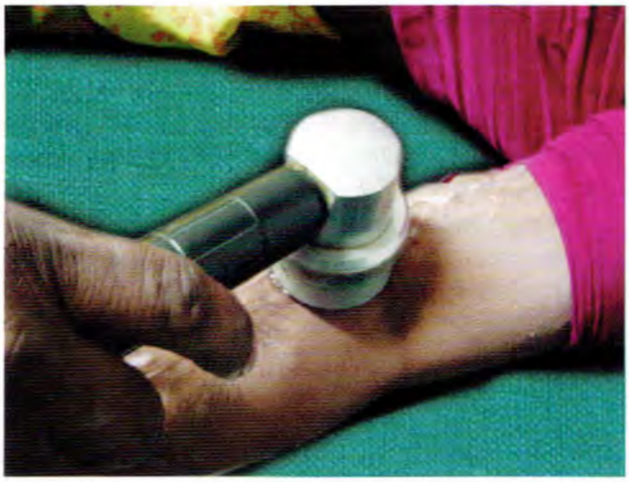


Fig. 33.12: Ultrasonic treatment in progress

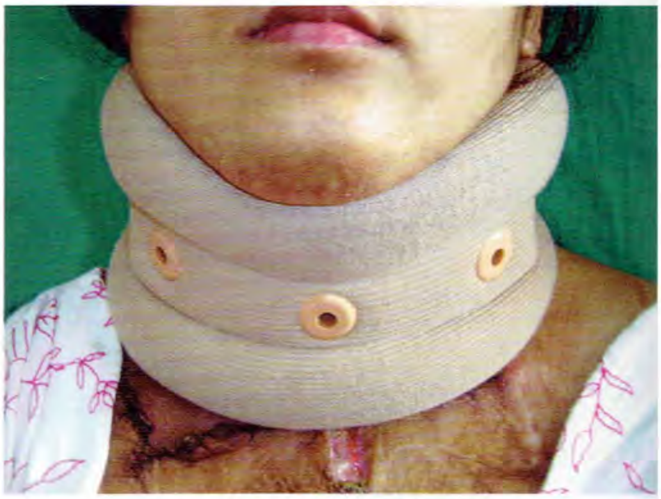


Fig. 33.13: Simple cervical collar

to the ultraviolet rays. The patient and the therapist must use dark glasses to avoid ultraviolet rays. The treatment is given for duration of 15-20 minutes followed by usual dressing. It gives good results and the patient is likely to cooperate more willingly during the dressings.

The ultraviolet lamp should be placed in the dressing room so that the patient is not required to move unnecessarily. It is recommended that the therapist goes to the dressing room to give the ultraviolet treatment so that she/he can correct the position of the particular body part, give exercises and tie the splint after the dressing.

Management of Grafted Neck Burn Wounds

The most common burn injuries to the neck are in the anterior region. In acute cases alone, the patient must be given cervical collar (hard) during the dressing so that the patient does not develop a flexion contracture. The position of the neck must be maintained till the wound healing is completed. After grafting of neck wounds, the patient's neck is kept in hyperextension to keep the graft in the stretched position (Fig. 33.13).

The exercises recommended are the same as those recommended in the acute stage.

The exercises are detailed as follows:

i. Ask the patient to press the head down towards the bed.
ii. No head movement must be allowed as these are only muscle-strengthening exercises.
iii. The same exercise should be repeated sideways. This makes the patient comfortable and gives a feeling of well-being.
iv. All the movement of shoulders and elbows and hands are done,
v. This is followed by leg movements to ambulate the patient as early as possible,
vi. It is very important to teach the patient how to get off the bed without disturbing the neck. The patient must first sit up in bed, then put the legs down and stand up. During this movement, the patient may feel giddy, nauseous for a few days but this soon normalises with the neck exercises.
vii. When the dressing of the grafted part and the donor part is removed, post grafting exercises and massage should be started.
    a. Patient should be put in supine position with neck in extension.
    b. Massage with coconut oil or some soft lubricant should be done.
    c. Efflurage massage followed by thumb kneading for 15 to 20 minutes. The patient should be taught how to massage so that he can look after the grafted part himself.
    d. Exercises of the neck should be followed by massage to keep the grafted skin soft and supple in the right position. Silicon gel may be applied after which pressure garment may be used to avoid hypertrophy of scars (Fig. 33.14).

Even if the patient complains of discomfort due to warm climate, the pressure garment and collar must be used and its importance explained to the patient.

In children four-post collar (Figs 33.15A and B) is advisable as they are growing and such a collar can be

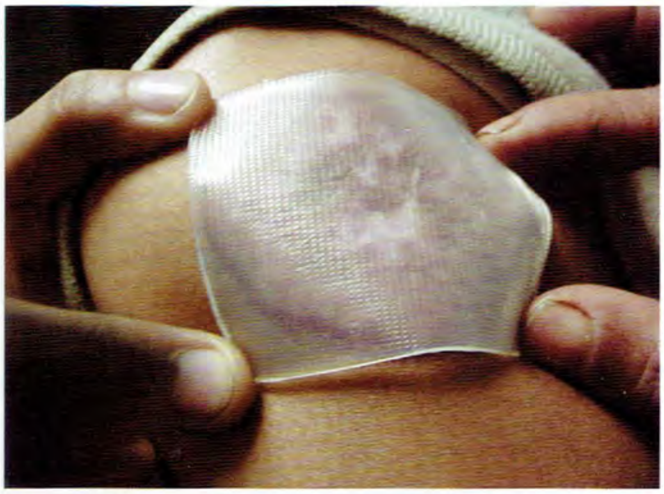
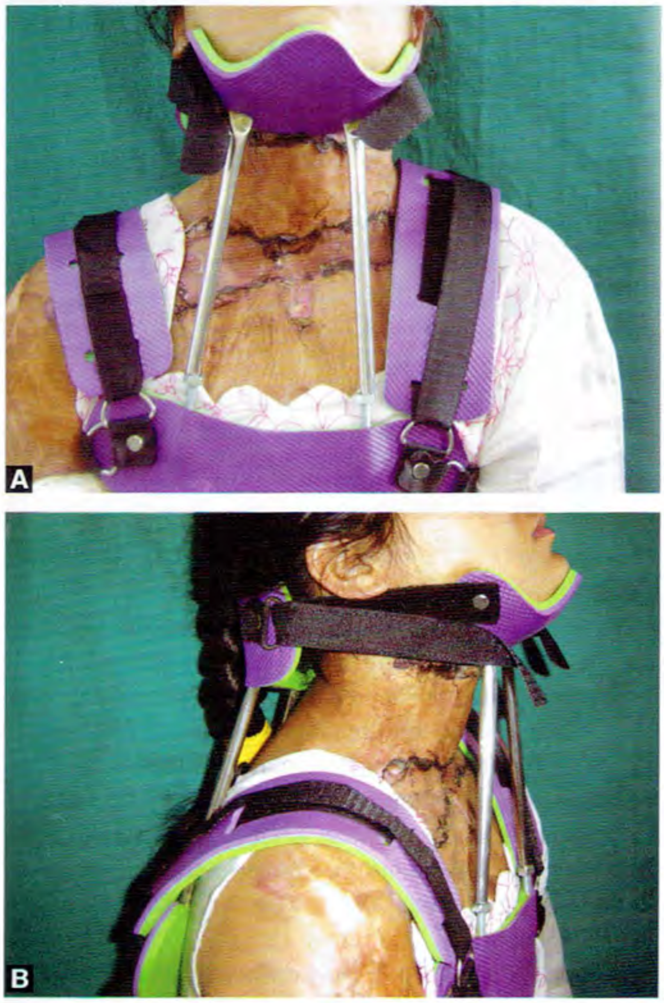


Fig. 33.14: Application of silicon gel sheet on hypertrophic scar



Figs 33.15A and B: Adjustable four-post collar

adjusted easily. It is crucial to ensure that the patient does not develop an ulcer because of an ill-fitting collar or pressure garment. When the pressure garment or collar is worn for the first time, the patient should be asked to remove it often and air the body part till he gets used to it.

The collar must be well-fitting and not too tight. The collar must be worn for six to ten months.

Massage should be continued for a year and must always be done with clean hands and nails. The use of a pillow must be strictly avoided for a period of one year.

Management of Grafted Axillary Burn Wounds

If the axilla has a deep burn, skin grafting is required. The graft has to be taken care of in the same manner as the neck (given above). Abduction splint is given to maintain the graft and prevent graft contraction (Fig. 33.16).

After the operation breathing exercises are given. The patient should be ambulated and he can initially use the other arm to support the operated arm. Elbow, wrist and hand exercises must also be done (Fig. 33.17). The patient must be given scapular adduction and elevation exercises. It is crucial that the attention be paid to the patient's posture so that he does not develop scoliosis. In children this is particularly critical as lack of care may lead to permanent scoliosis. Once the bandage is removed and the graft is in place, exercises of the shoulder must be started immediately (Fig. 33.18). This is crucial as the shoulder may become stiff due to immobilization in the cast. In children, mobilization of the shoulder takes place relatively early as compared to adults who may suffer from frozen shoulder

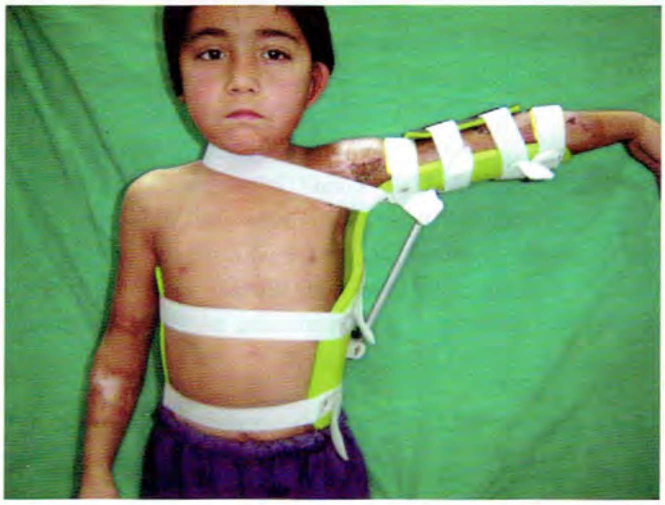


Fig. 33.16: Abduction splint

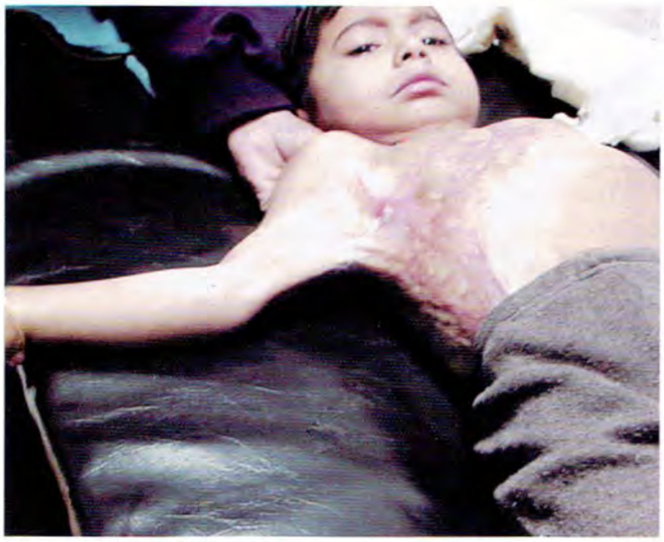


Fig. 33.17: Axillary contracture

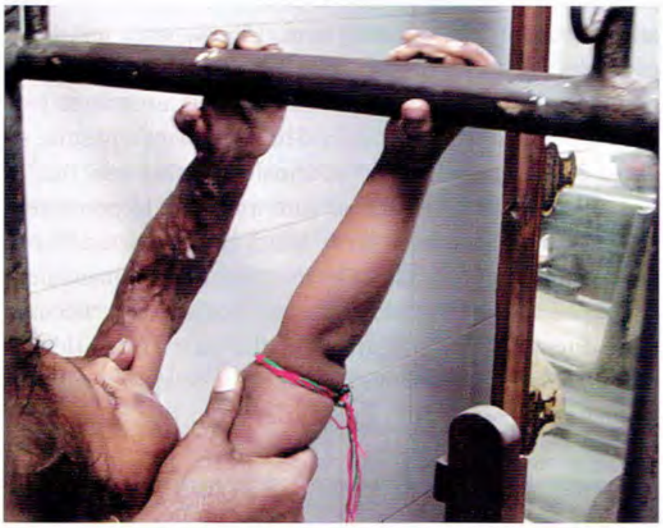


Fig. 33.18: Shoulder exercises in children after correction in axillary contracture

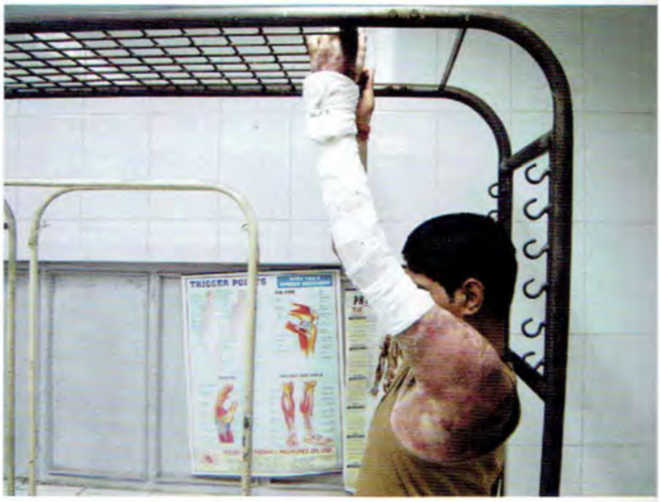


Fig. 33.19A: Shoulder exercises

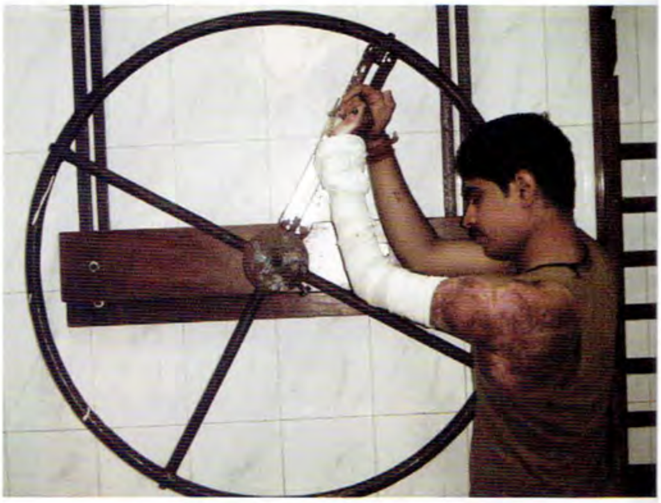


Fig. 33.19B: Equipment for physiotherapy of shoulder joint

or periarthritis (Figs 33.19A and B). In such cases the patient may be given Interferential Therapy (IFT) to reduce the pain followed by exercises. If the pain is local, ultrasound therapy may be combined with the exercises for speedy recovery. The patient should also be taught home-based exercises to get effective results.

Management of Grafted Elbow Burn Wounds

Grafting on elbow wounds may be of three types:

1. *Elbow grafting on anterior surface:* If the grafting is done only on the *anterior* surface, the elbow will be in a straight position and an anterior splint will be provided. The patient should be given shoulder exercises with support along with hand exercises. As soon as the graft is taken up and the bandages removed, massage is initiated. Elbow exercises within the tolerance level of pain of the patient may also be started. Passive movements of the elbow are not indicated as the chances of myositis ossificans are greater. After massage and exercises, the patient should be given pressure garment and splints (Fig. 33.20).
2. *Elbow grafting on posterior surface:* If grafting is done on *posterior* side, the elbow is kept slightly in flexion. Splint support is not required. A regular schedule of

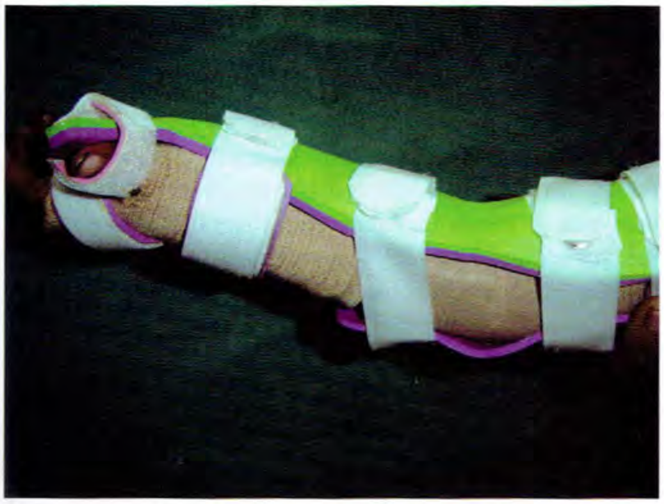


Fig. 33.20: Extension splint at elbow

massage, exercises and use of pressure garment after graft take are adequate to help the patient.

3. *Elbow grafting circumferentially:* In case of *circumferential* burns, the splint used is the same as in the case of anterior burns. It is important to ensure that exercises are given with great care as these may be very painful for the patient. This is also crucial to ensure that the graft from the extensor side is not stretched too much in extreme flexion.

It is recommended that active exercises of the elbow are taught with flexion, extension, supination and pronation in the sitting position, with the humerus well-supported and with a dumbbell in hand.

Management of Grafted Wrist Burn Wounds

Wrist burns can result in four types of contracture if not grafted in time:

- Wrist flexion
- Wrist extension
- Ulnar deviation
- Radial deviation

In the case of grafting on the *flexor surface of wrist*, the postoperative hand should be elevated to reduce edema. After the graft take, exercises are started and only extension must be encouraged for the initial 2-3 days followed by finger flexions. Cock up splint is given to maintain wrist extension.

After the dressing is removed, massage should be done using coconut oil and the patient should be encouraged to use the hand. This is crucial to avoid graft contracture and facilitate maximum functioning of the hand. The use of a splint helps to keep the wrist in extension.

The patient must wear the pressure garment for 6-12 months. In case the patient desires to use the injured hand for household work such as cooking, the pressure garment may be removed for that period of time and put in use again immediately after the task is over. This is essential to keep the graft in the correct position and avoid any hypertrophy of the scar.

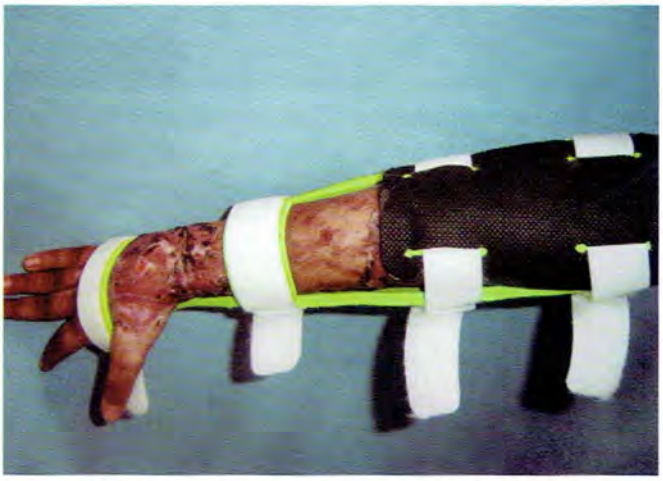


Fig. 33.21: Wrist and elbow contracture released

When a graft is applied on *extensor surface of wrist* the hand must be kept in a straight position and elevated (Fig. 33.21). Movement of the fingers must be encouraged after graft take. When the dressing is removed and the graft has healed well, the massage and finger exercises can be initiated. Along with wrist movement given by the therapist, the patient must be encouraged to use the hand for normal functions. A pressure garment and splint must be used at night. A considerable amount of wrist flexion must be given to ensure that the patient does not get wrist extension contracture.

If the graft is applied on the *ulnar* side, the hand must be stretched on the radial side to keep the graft stretched. Similarly, if the graft is on the radial side, the hand should be stretched on the ulnar side. In both cases hand movements must be continued along with the use of splints and pressure garments for a prolonged period of time.

Management of Hand Burns

Hands are very prone to burns as they are instinctively used to protect the body from any physical harm. Similarly hands are the most used parts of the body for all tasks. As a result, burn injuries of the hands leave the patient extremely dependent for even routine activities. Although they are relatively small body parts, hands play a crucial role in major body functions. Hands comprise of small inter-articulating bones, muscles, blood vessels and nerves covered with skin. To cater to different functions, the hand is covered with two different types of skin: Dorsal and Palmar. The Palmar skin is very thick and can handle rough objects whereas the dorsal skin is thin and supple.

An understanding of the structure of the hand and its parts is crucial in determining a program for its rehabilitation. The hand complex consists of five digits each of which has a metacarpal and a metacarpophalangeal (MCP) joint. Each finger has two interphalangeal (IP) joints, while the thumb has only one. There are 19 bones, distal to the carpals and 19 joints that together make up the hand complex.

Articulations in hand are of three types, viz., carpometacarpal, metacarpophalangeal and interphalangeal joints. The muscles of the hand are classified as extrinsic and intrinsic muscles. These muscles join together to perform the function of the fingers and the wrist.

Extensors of forearm comprise of:

- Brachioradialis;
- Extensor carpi radialis longus
- Extensor carpi radialis brevis
- Extensor indices,
- Extensor digitorum communis,
- Extensor digiti minimi,
- Extensor carpi ulnaris
- Anconeus.

Flexors of the forearm and hand comprise of:

- Pronator teres
- Flexor carpi radialis
- Palmaris longus
- Flexor carpi ulnaris
- Flexor digitorum profundus
- Sublimis

Intrinsic muscles of hand are:

- Thenar group
- Hypothenar group
- Lumbricals
- Interossei

The wrist performs the function of flexion, extension, ulnar and radial deviation and circumduction. The thenar group of muscles perform the thumb functions; hypothenar group of muscles perform the function of the little finger while the interossei and lumbricals perform abduction and adduction of the fingers. These combine with extensor tendons for finger extension.

All the above muscles are responsible for different functions for the hand. They are attached to small bones in the hand. The hand also consists of some important soft tissues that help in different hand functions. For instance, the volar plate is a multi-layered structure consisting of fibrocartilage, responsible for thickening of the inside of the joint capsule.

The joint capsule, superficially located transverse metacarpal ligament and transverse fibers blend with original longitudinal fibers of the MCP joint capsule to get co-ordinated action of the MCP joint. Flexible attachment of the plate to the phalanx permits the plate to glide proximally along the volar surface of the metacarpal head without restricting motion. This fibrocartilage mechanism also prevents pinching of the long flexor tendons during flexion. In the case of extension, the plate adds to the amount of surface in contact with the large metacarpal head. The plate also helps to restrict the hyperextension that is permitted by the loose capsule.

The position of comfort is always preferred by the patient and this position predisposes to contracture formation, e.g. in the hand, the typical post burn claw hand results from keeping the hand in position of rest, i.e. wrist flexed, MCP joints extended and IP joints flexed. It is important to avoid this position while dressing the areas. Elevation of burnt hands is also essential to reduce edema. Active exercises are encouraged by the use of open method of treatment. In this, the hand is smeared with silver sulfadiazine cream and enclosed in a polythene bag in which movements of all the joints are allowed and also easily monitored.

In case of hand burns, the following deformities are possible:

- Typical claw hand
- Boutonnière deformity
- Swan neck deformity
- Subluxation of MP joint
- Mallet finger

Typical claw hand is characterized by MCP joint extension, IP joint flexion and thumb adduction (Fig. 33.22).This deformity results in a loss of grip and pinch resulting in nearly 60% loss of hand function. This contracture, can be avoided if splinting is done from the very beginning followed by active exercises. It is critical that no passive movement is given in acute stage to avoid rupture of any soft tissue.

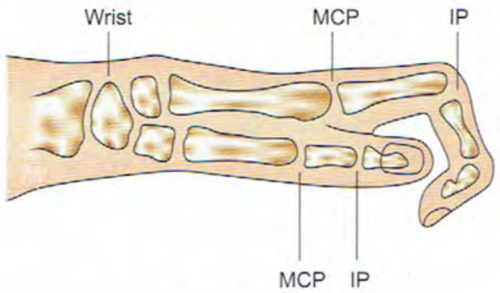


Fig. 33.22: Typical claw hand

Different types of splint may be used to avoid this deformity (Figs 33.23 to 33.24B). A *Simple Static Splint* provides flexion at the MP joint and extension at the IP Joint. The thumb is maintained in abduction. Once the grafting of hand is completed, the patient may be given a pressure garment and a splint to maintain the correct position of the hand. Exercises must be done 2-3 times a day (Fig. 33.25). Once the area has healed, the patient must be encouraged to use the hand ensuring no further injury. If a child has received this treatment, he must be encouraged to attend school and perform his daily tasks himself.

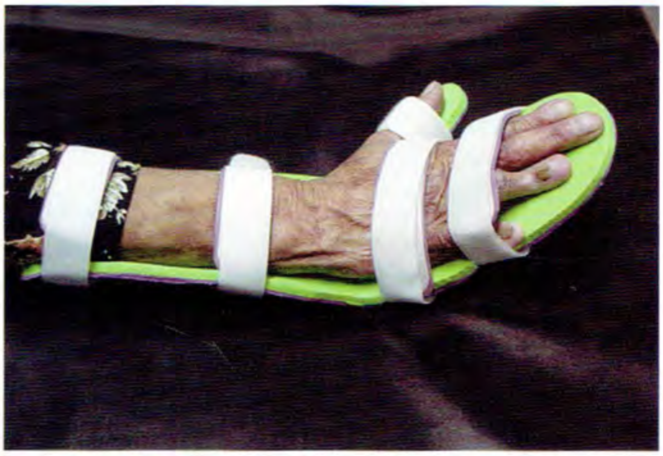


Fig. 33.23: Cock-up splint

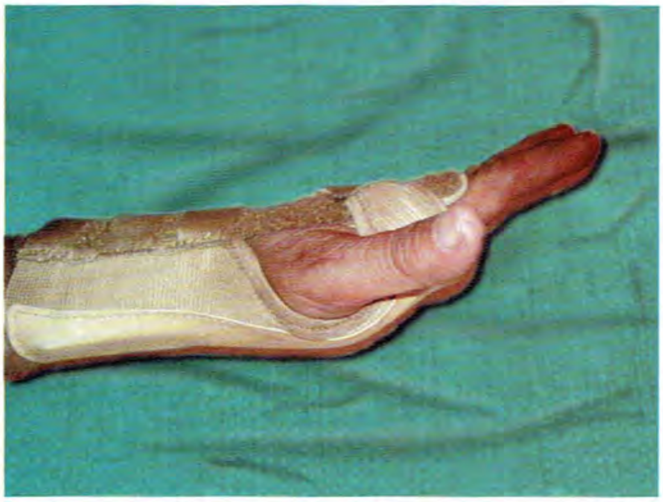


Fig. 33.24A: Short Cock-up splint (Functional cock-up splint)

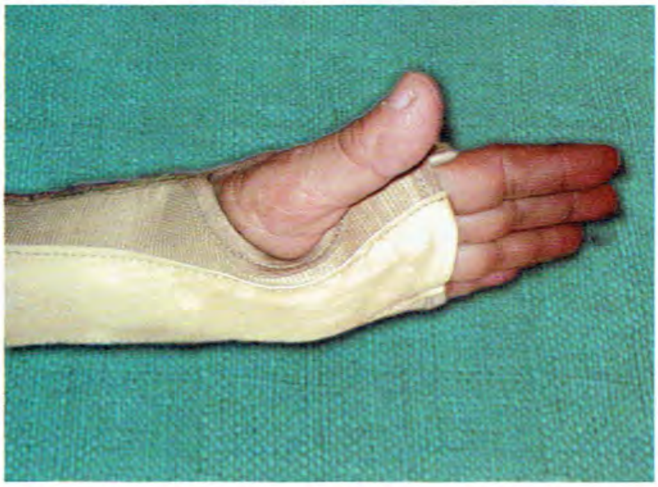


Fig. 33.24B: Same splint–Palmar view

Boutonnière Deformity

A deformity of this nature involves a stretch or tear of the central extensor slip, as a result of which the lateral bands migrate volarly and flex the proximal IP Joints (Fig. 33.26). The distal phalanx also extends due to stretching of the lateral bands. The net result is that the intrinsic and extensors migrate proximally, resulting in this particular deformity. A mild deformity does not affect the functioning of the hand but if it is severe, the patient is unable to tighten the fist and also loses pinch movement.

It is recommended that finger splints be given at an early stage to avoid stretching of the lateral slip. The required splints, for individual fingers, are easily available commercially. The same splint can be used as either static or functional splint. However, if all the fingers are involved, a simple Cock-up Splint is recommended.

Swan Neck Deformity

The contracture of the intrinsic muscles causes flexion at the MP Joint, hyperextension at the proximal IP Joint and flexion at the Distal IP Joint. This deformity can also be avoided by the early use of a splint and correct timely exercises. If contracture has already taken place and the capsule is damaged, it is crucial that surgery is followed by splinting and exercises (Fig. 33.27).

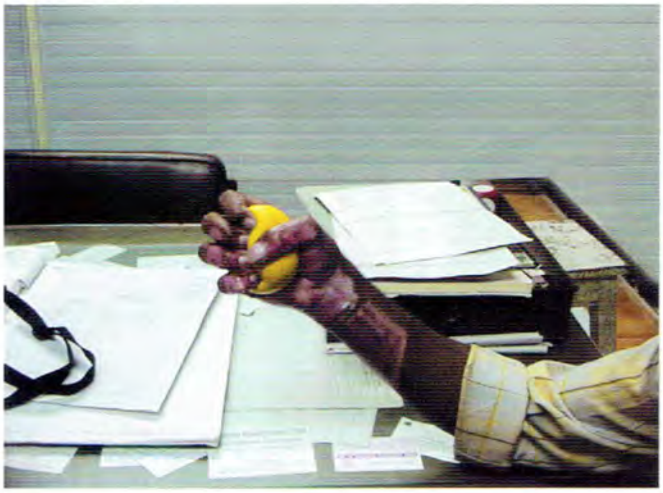


Fig. 33.25: Hand exercises after correction

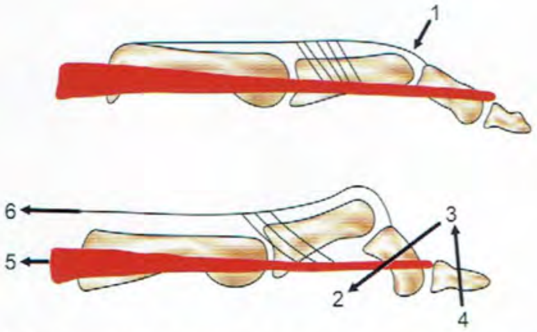


Fig. 33.26: Typical boutennière deformity. Arrows 1-6 showing the direction in which the force on bones and joints acts to cause the deformity

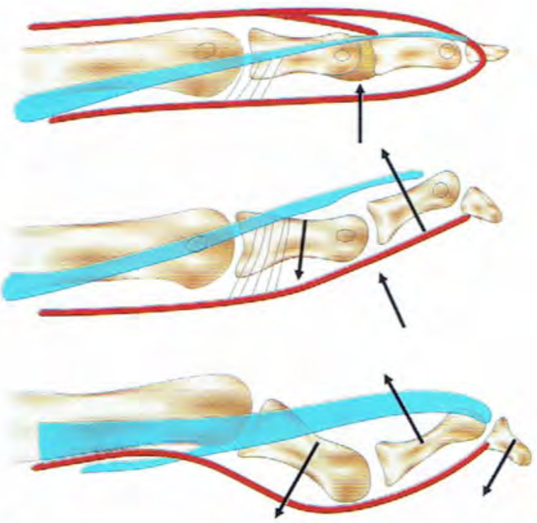


Fig. 33.27: Typical swan neck deformity subluxation of MP joint. Arrows showing the direction in which bones and joints get deformed

Such a deformity may involve the tearing of the capsule or herniation of the head of the metacarpal or the base of the phalanx. Extension of the MP Joint may affect the grip of the hand (Fig. 33.28).

The rehabilitation of hand consists of splinting the MCP joint in flexion using a static splint initially, followed by the use of a functional splint for a few weeks accompanied by massage and exercises. Active exercises are recommended and the patient must be encouraged to start performing his normal tasks as early as possible.

Mallet Finger

This is caused by a rupture or tear of the extensor tendon, proximal to the insertion of the lateral band on the proximal aspect of the distal phalanx. The resultant damage allows the horizontal proximal pull of the extensor tendon and flexor digitorum profundus tendons, causing a loss of extension of distal phalanx (Fig. 33.29).

This deformity can be a deterrent to minute hand functions. It may be corrected surgically followed by splinting and exercises.

Functional splints for different hand and finger deformities are available in different sizes (Figs 33.30A to H).

Management of Burns of Lower Extremities

There are various deformities which can occur following burns of lower extremities.

- Flexion contracture of hip
- Hip adduction and perineal contracture
- Flexion contracture of knee

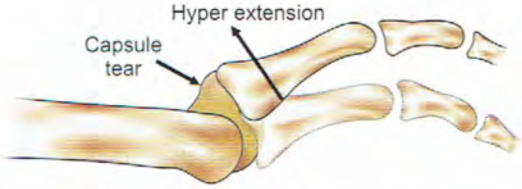


Fig. 33.28: Subluxation of MP joint

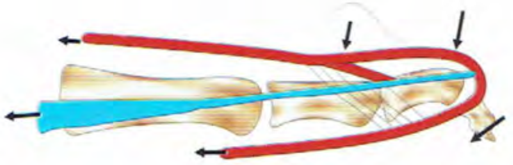
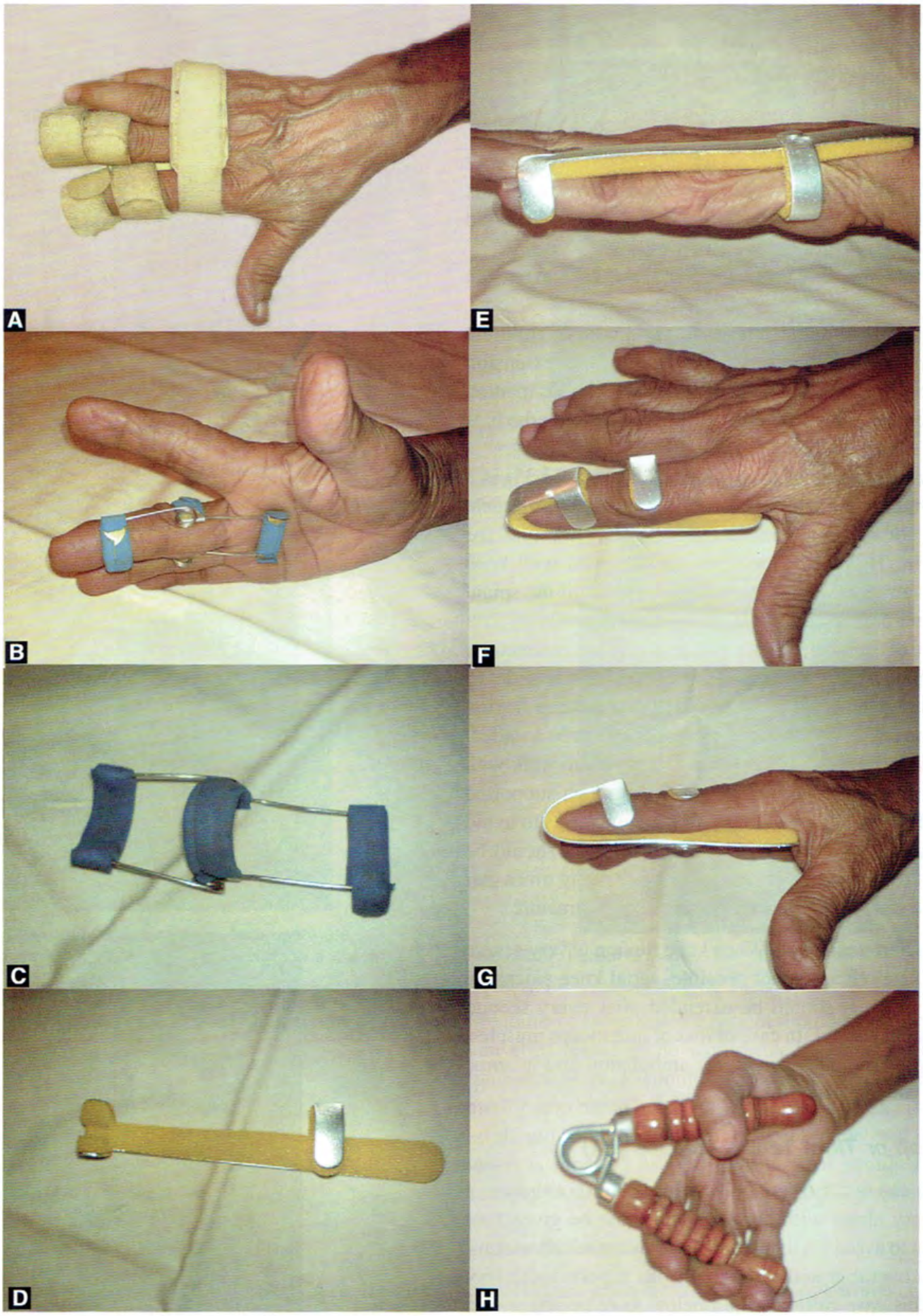


Fig. 33.29: Mallet finger. Arrows showing the direction in which the force on bones and joints acts to cause the deformity



Figs 33.30A to H: (A) Individual Finger Splints (B to G) Individual finger splints (Readymade Splints available). (H) Spring for improving grip

- Foot drop
- Calcaneal deformity
- Varus deformity
- Valgus deformity
- Flexion contracture of hip—Comfortable position is the key to hip flexion contracture (Fig. 33.31). Whenever there is abdominal burn or hip burn, patient tries to flex the knee and hip to make himself comfortable. To avoid this contracture, there are two positions.

a. Keep a hard pillow under the pelvis and knee extension splint bilaterally.
b. Keep the patient in prone position. In case of children, hip spica (Figs 33.32 and 33.33)with knee extension can be given till the wound is healed and graft is mature.

Perineal contracture can be avoided by keeping the hips abducted.

Flexion contracture of knee (Figs 33.34 and 33.35) is a very common contracture because it is a very comfortable position for sleeping and sitting but is very bad for ambulation. The patient becomes handicapped with knee flexion contracture. If not corrected, it can spoil the spinal posture and patient may develop kyphosis.

In case of a child with unilateral knee flexion contracture, patient may develop contracture on normal side to compensate shortening of affected leg due to knee flexion contracture and may develop scoliosis. This is to be avoided. If grafting is done, the patient should be made to walk with the support of crutches for a few days and then supported with a splint. Patient must be given knee exercises to avoid any weakening of muscles (Fig. 33.36). Patient should be given gait training after the exercises since many times gait is affected due to prolonged knee flexion contracture.

Splinting and grafting—When knee flexion is very severe and complete release is not possible, serial knee extension splint is given. It should be extended after every second day if it is not painful. In case of loss of quadriceps muscles, knee support should be given for ambulation and in some cases by calipers (Fig. 33.37).

Foot Drop or Tight Tendo Achilles (TA)

Foot drop can occur if tibialis anterior is burnt or there is a nerve injury along with burn. Patient must be given foot drop splint to avoid TA tightness. Sometimes the nerve injury is non-recoverable and the patient has a permanent foot drop. In this case caliper with a below knee posterior stop at ankle is given. This enables the patient to walk easily without lifting the hip to clear the ground. If a caliper is not

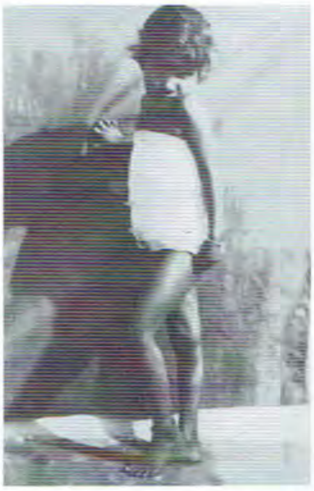


Fig. 33.31: Patient with hip flexion contracture

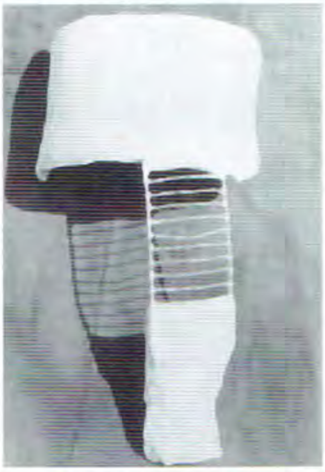


Fig. 33.32: Hip spica

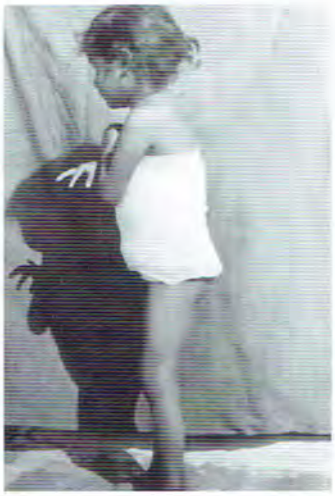


Fig. 33.33: Hip correction after use of hip spica

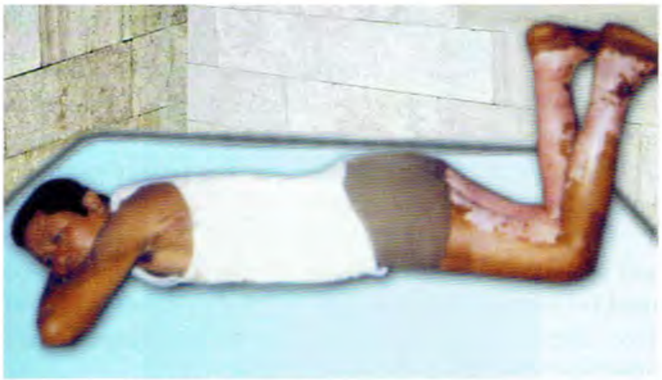


Fig. 33.34: Bilateral knee flexion contracture

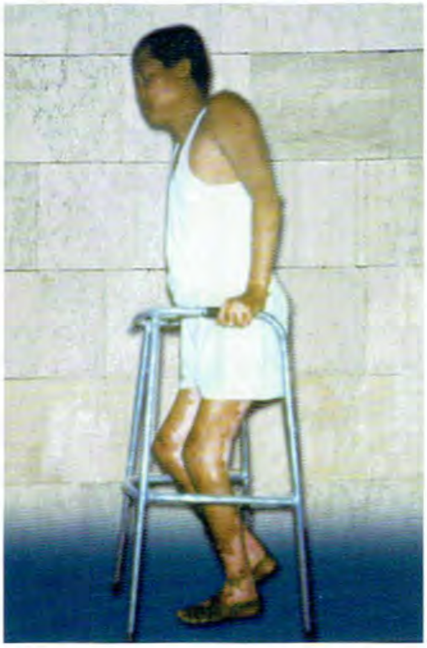


Fig. 33.35: After serial splintage, both knees are corrected and patient is made to ambulate with a walker

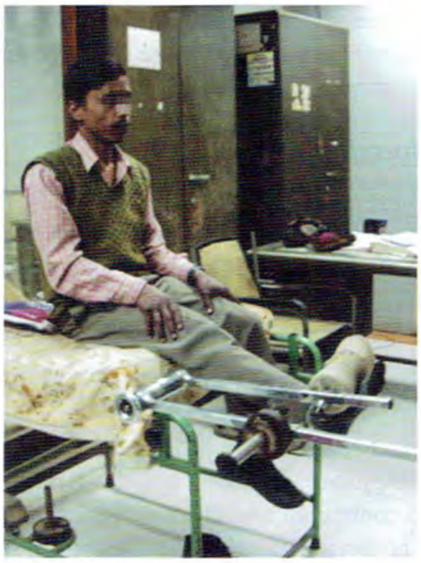


Fig. 33.36: Quadriceps knee exercises after the correction

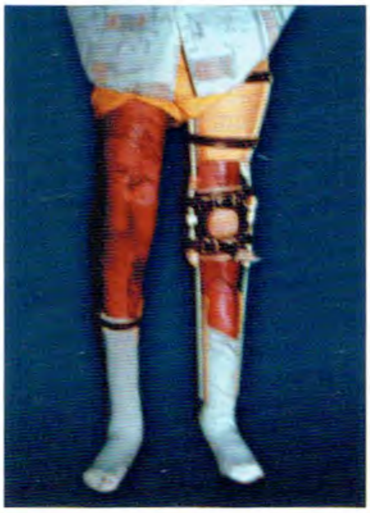


Fig. 33.37: Patient is made to ambulate with above-knee caliper

given, the patient will walk raising the hip and may develop the following problems:

- Backache
- Wrong gait
- TA tightness
- Inability to turn
- Early exhaustion

Calcaneal Deformity

Calcaneal deformity occurs when **tendo Achilles or gastrocnemius** is burnt and not repaired. Patient will not have heel to toe gait and will develop hyperextension at knee joint. To avoid this, the patient should be given a caliper even after repair as he would need support to heal the repaired tendon. It should be a below knee caliper with anterior stop bar to avoid powerful foot dorsiflexion. Heel-pad should be given to get proper heel to toe gait. Once the tendon is repaired properly, the patient should be given exercises to strengthen TA and proper gait training.

Valgus Deformity

Valgus occurs when there is tightness of Peronei and tibialis anterior muscles due to burn. Here tendon repair is required followed by a caliper to ambulate the patient and avoid recurrence of valgus deformity. If deformity occurs again, then for one year the patient is given a caliper with a below knee inside t-strap to avoid valgus position.

Varus Deformity

If peronei muscles are lost due to deep burns, flexors and tibialis anterior are very strong muscles and foot goes in varus position. To avoid this, repair may be needed and then the patient has to be given exercises to strengthen peronei and below knee caliper for some time is indicated.

TYPES OF SPLINTS

After the initial resuscitative procedures brings the patient out of the acute phase, all efforts have to be made to prevent burn wound contractures in order to minimize deformities later on if no action is taken in the initial stages. The fabrication and application of splints is of utmost importance. Splints maintain the joints in anti-deformity position and each splint should be ideally custom fitted for every individual patient.

There are 3 kinds of splints used in burn patients–Static, dynamic and semi-dynamic.

A *static* splint keeps the part in a particular position and does not allow movement of that part.

Dynamic splint supports that part but allows movement.

Semi-dynamic splint supports the part and allows very little movement. It all depends on the condition of the patient.

Splint Selection

Nowadays, readymade splints are available in all sizes; yet sometimes, tailor made splint may be needed. Splints should be light, well padded, easy to wear and easily removable. Materials used in splints should not be toxic, should be easily available and economical. The material should be non-absorbent to avoid bacterial growth. Expediency is essential in the fabrication of splints. Esthetics should play a secondary role. With no other treatment, there is a higher occurrence of deformity that may ultimately require many surgical procedures and an extended period of rehabilitation.

Thermoplastics are easy to use and make a static splint. It can be used for 'serial' splintage as it can be reused after putting it in hot water. It is advisable to cut different types of cock-up splints and knee extension splints so that it does not take much time to create a splint.

Dynamic splints are readily available and can be obtained for all body parts – fingers to axilla and foot to knee. They are very easy to use and do not restrict joint ranges or muscle power.

Pressure Garments

After the wounds have healed, pressure garments help to reduce thickness of scars and flatten irregularly thickened areas thus preventing hypertrophic scars and keloids. Though all scars improve with time, the pressure garments help to accelerate the process by early maturation of scars.

Pressure garments can be readymade or tailor-made to suit specific needs. Readymade pressure garments can be used for parts like extremities, chin and wrist but in case of face, chest and perineum only tailor-made garments are effective(Figs 33.38 to 33.40).

In case of lower extremity, after the grafting, a patient, if made to stand straightway, will feel pin pricks and his graft will develop blistering because of venous congestion. To avoid this, the patient should be taught dependency exercises and made to wear pressure garments.

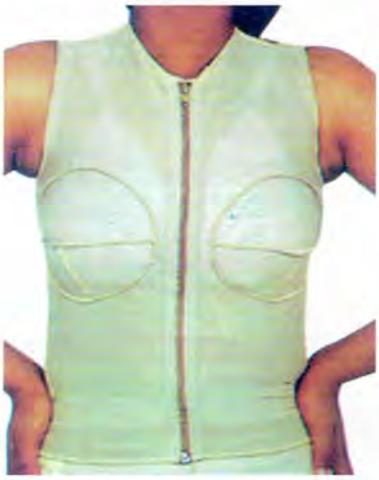


Fig. 33.38: Custom made pressure garment for full body (*Courtesy:* Technomed Pvt. Ltd.)

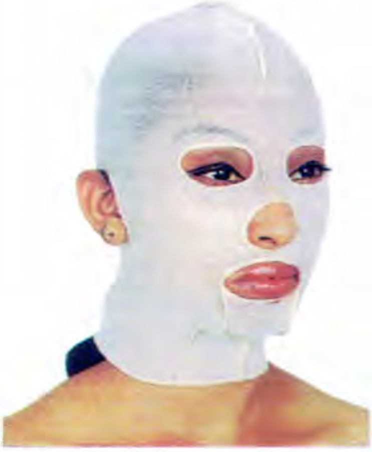


Fig. 33.39: Pressure garment for face (*Courtesy:* Technomed Pvt. Ltd.)

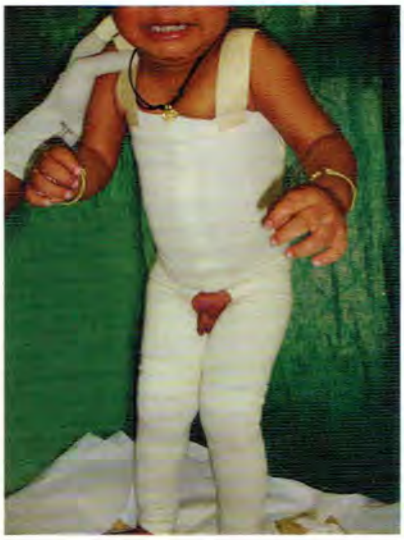


Fig. 33.40: Tailor made whole body pressure garment in a child

Dependency Exercise

The patient should be made to do dorsi flexion and planter flexion with high sitting and then patient should be given quadriceps exercises and made to stand and walk for one minute. Gradually time duration should be increased and within a few days, the patient will feel normal.

Duration of Use

Pressure garments should be used for 6 months to a year. Initially, the patient must use pressure garments for 22 hours with only minimum time for personal hygiene and exercises. After 6 months the patient should use the garments only at night. This will help keep the graft in the right position.

Quality and Maintenance

The material used for pressure garments should be skin friendly (non-allergic), breathable (porous), equally stretchable on all sides, should be tested for transfer of pressure on skin and should be constructed according to venous pressure gradient. It should be certified as a medical device by an independent authority of the respective country like CE or FDA approved.

The garments should be cleaned everyday with soap and cold water. They should not be dried in the sun. Pressure garments should be well fitting and easy to remove. They should have breathing holes.

General Physiotherapy

Apart from providing care to prevent stiff joints, a physiotherapist needs to ensure that the chances of chest complications, bed sores and deep vein thrombosis in these chronic bed ridden patients especially the elderly age groups are minimized. For this chest physiotherapy and postural drainage, spirometer exercises need to be administered to the patient daily. To prevent deep vein thrombosis, the patient should be ambulated as soon as possible or if patient is unable to walk, he/she should be made to sit and move his/her limbs.

For postural drainage, either rotating beds (Stryker frames) are used or it is done manually by the nursing staff. Air mattresses or water beds should be used to prevent bed sores with 1-2 hourly change of posture.

Deep breathing exercises should also be encouraged for proper expansion of lungs.

SUMMARY

Intensive physiotherapy of burned patient should begin from the first post burn day and continued not only till the patient's wounds heal but also beyond that till full function of all joints is restored. The treatment program should include daily evaluation, supervision, repeated exercises and splintage to achieve the best possible results. The improvement in joint movement and muscle strength and prevention of deformities by physical therapy and rehabilitation will help the patient to become functionally independent.

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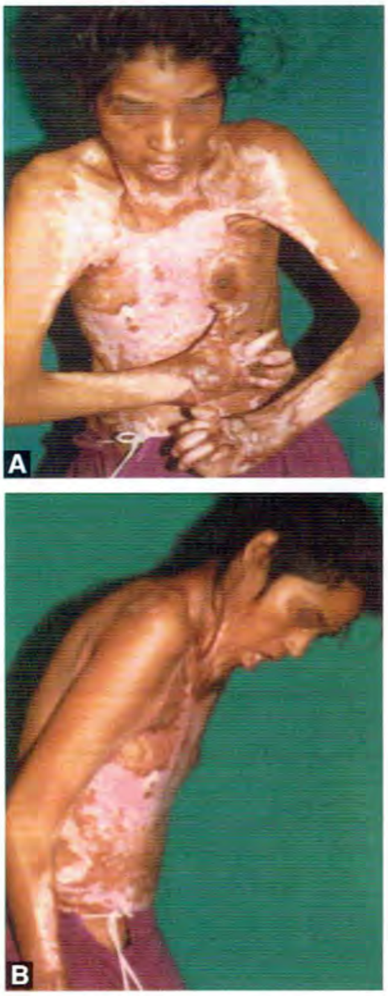
Chapter 34

Post Burn Sequelae and their Management

Arun Goel

A severe burn injury is the most devastating injury a person can sustain and yet hope to survive! These injuries constitute the second commonest cause of all trauma-related deaths in the developing as well as the developed countries. Over the last sixty years, great advances have been made in the field of burns at basic as well as applied level. The pathophysiology of burn injury has been understood at cellular as well as molecular level in great detail. Treatment methods have been devised to counter and reverse these changes. Burns are now being treated scientifically in a well standardized manner. The mortality rates and the morbidity from burns have diminished greatly so much so that a mere survival of an extensively burnt patient (say, 65-70% Total Body Surface Area (TBSA)) is no longer considered a great outcome. As is true for any other life threatening injury, survival is the immediate concern for the burn patient, his/her relatives and the treating clinician. Actually, it is the degree of restoration to pre-burn level and 'return to society' which is more important. Burn survival statistics can indeed be misleading! Unfortunately, the tremendous advancements in the field of burns have not benefitted a very large majority of burn victims in India and other developing/under-developed countries. Post burn sequelae resulting in disfigurement with grotesque appearance and deformities with crippling contractures/amputations continues to be a great challenge to the modern burn surgeons in their attempt to restore the burnt patient to pre-injury status (Figs 34.1A to D).

As recently as 1986, Brown and Barot[1] wrote, "The ultimate goal in the treatment of burns is the establishment of a closed wound with restoration of the intact cutaneous barrier protecting the patient from his or her environment."



Figs 34.1A and B: Front and lateral view of a 28-year-old female with extensive post thermal burn scars, severe and multiple contractures of neck, axillae, wrists and hands. Also note the kyphotic posture and loss of right nipple-areola complex

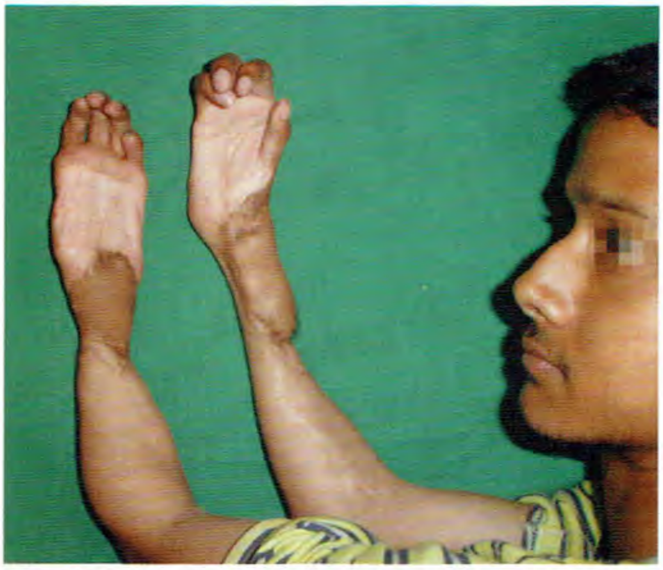


Fig. 34.1C: A 16-year-old male with post electric contact burns bilateral hand deformity with loss of digits and flexor tendons. Distant flap covers done to perform reconstructive surgery

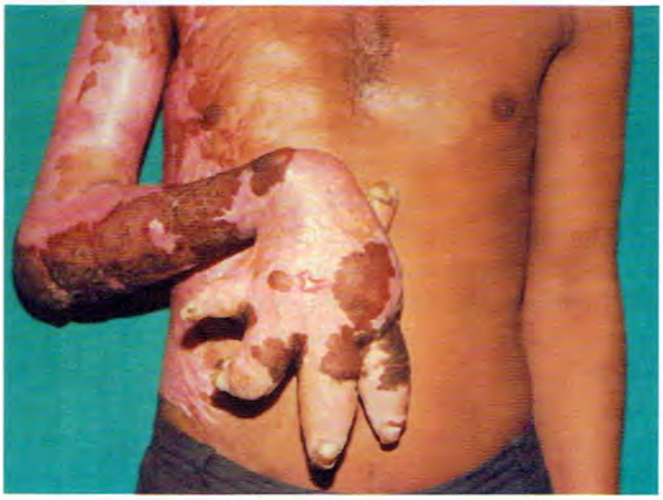


Fig. 34.1D: Grotesque deformity and loss of function of right hand in a 30-year-old male

The present author would like to replace the word "ultimate" with "immediate". Unfortunately, such a healed burn patient may be left with one or more of the various sequelae in different permutations and combinations. These post burn sequelae have varying degrees of functional and aesthetic components. The actual prevalence of these disabling conditions in general population can never be gauzed accurately, especially in a vast country like ours. However, it must be inversely proportional to the standards of initial treatment with patients receiving best of care having minimum number and severity of these sequelae.

For descriptive purposes, the various post burn sequelae have been considered under following headings. An individual burn patient may have any number of them in varying severity. Surgical reconstruction[2-5] of these sequelae requires the knowledge and expertise of the full spectrum of a plastic surgeon's armamentarium from local excision, serial excisions, tissue expansion, skin grafts, local flaps, regional flaps, free flaps and prefabricated flaps.

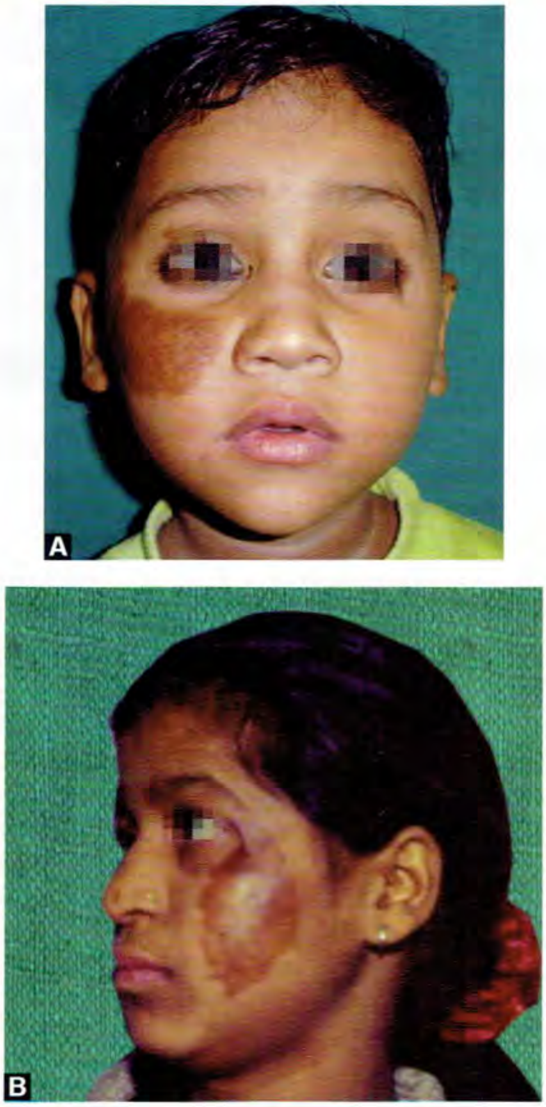
1. Post burn scars,
2. Post burn contractures,
3. Post burn losses,
4. Chronic unhealed areas,
5. Bone and joint deformities,
6. Development of malignancy,
7. Miscellaneous and
8. Psychological disorders.

POST BURN SCARS[6-10]

Post burn scars are inevitable even with the best of treatment. This is because they depend upon the depth of burn injury. Healing of the burnt skin is a complex process where nature tries to close a wound by deposition of collagen and re-epithelialization to restore the continuity of the skin. In superficial dermal burns which heal in less than 14 days, the healing may be so perfect as to resemble the normal skin and become indistinguishable from the surrounding unburnt skin. All other deeper burn injuries lead to healed "areas" which are clearly different from the surrounding unburnt skin with respect to their color, texture, dullness/ shininess, stretch ability, hair pattern, thickness etc. even when the healing had proceeded "normally" (Figs 34.2A and B). In these cases and in all other cases where healing has not proceeded normally due to one or more of the various factors affecting wound healing adversely, the final appearance of the healed burnt areas is definitely different from the surrounding unburnt skin.

It must be remembered that the term "scar" has a different connotation for the plastic surgeon and the patient. The appearance of even the best skin grafted areas and the perfectly healed donor sites of these grafts is also a "scar" by the patient's definition of a scar.

The only way to erase the post-burn scars completely is to prevent the burn injury itself. 'An ounce of prevention is better than a pound of cure' is no better exemplified than by the burn injury. Once burn injury has occurred, the scars can only be minimized and not prevented or removed completely by appropriate patient and wound management in the acute phase including first aid and various physical



Figs 34.2A and B: Well healed post burn scars on face. Note the difference in color, texture and shininess. No deformity or functional deficit. Only esthetic considerations.

therapy measures instituted and religiously followed after the burn wound has healed completely till the complete maturation of the scar.

Bringing down the temperature of the skin to normal body temperature in thermal burns as soon as possible by pouring water minimizes the depth of burn injury. Similarly, depth in chemical burns can also be minimized by diluting the chemical thoroughly at the earliest (by water, in general). Radiation burn injury to skin in cancer patients has been minimized by improved technology.

Immediate correction of hypovolemic shock and hypoxia, not only benefits the patient as a whole but also allows for adequate perfusion of burnt tissues, especially in the zone of stasis.[11] The desiccation of the burn wound, increased viscosity of the blood and hypoxia of the patient all lead to death of compromised tissues which could have survived if appropriate therapeutic measures were instituted promptly. Along with infection, they can convert a burn wound into a deeper injury leading to more scarring. In addition, trauma to regenerating neoepithelium must be prevented by delicate handling of tissues and use of non-stick paraffin gauge and antimicrobial ointments/creams and moist dressings.

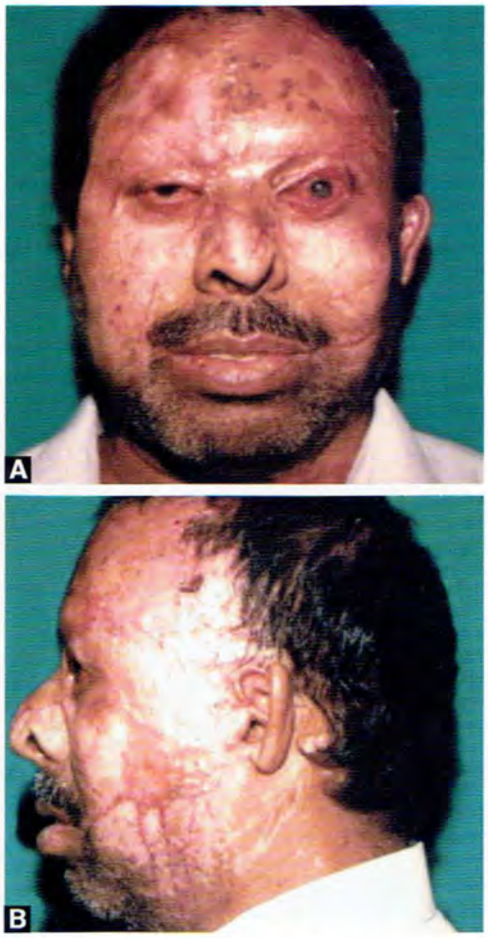
It must be noted that for a given depth of burn, the scars formed are not only different in different parts of the body but also different individuals show different outcomes for a given site and depth.

Again, for purely descriptive purposes, the completely healed burn patients may be divided into two categories depending on the time of their presentation to the specialist burn surgeon:

a. The patient is being treated by the burn surgeon from the acute stage and/or the scars are immature, and
b. The scars have already matured at the time of presentation to the burn specialist.

When the scars are immature (Figs 34.3A to C, 34.9A and B, 34.14A to E), their maturation can be altered favorably by using a number of physical therapeutic measures. These scars must not be exposed to sunlight till their color returns to normal or they have matured. The exposure to sunlight leads to hyper pigmentation of the scars and recently applied grafts. This is extremely difficult to treat. The exposure can be prevented by coverage with clothes, avoidance of outdoor activities, use of caps, pressure garments and sun screen lotions with SPF (Sun Protection Factor) of at least 20-25. The patients must be fore-warned on the use of various proprietary creams containing steroids for itching which is a common complaint in healing and recently healed burns. This is because steroids are photo-sensitizers leading to hyperpigmentation. When the scars are accompanied by contractures, deformities, etc. surgical interventions are planned to take care of all the problems together as detailed later.

When the scars are mature, soft and supple without any contracture, deformities etc. physical therapy measures cannot alter their appearance (Figs 34.2A and B). These patients need treatment purely to improve the esthetic appearance and regain the self-confidence. These are the patients who need the full spectrum of a plastic surgeons' armamentarium (Figs 34.4A to D, 34.25A to 34.27E and



Figs 34.3A and B: A 45-year man with healed homicidal acid burn injury with ectropion of eyelids, hazy corneas and immature facial scars

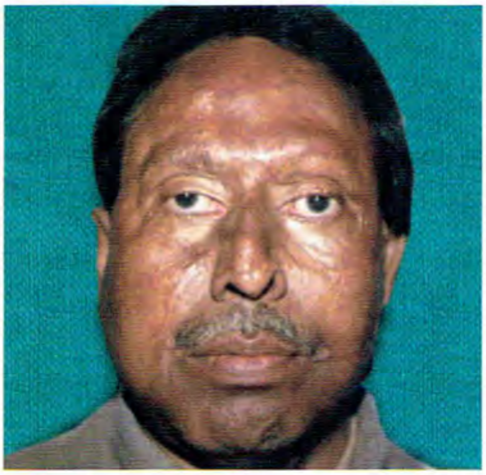
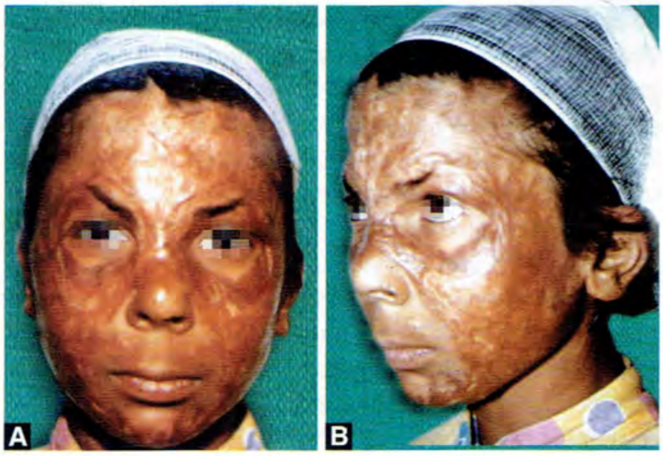
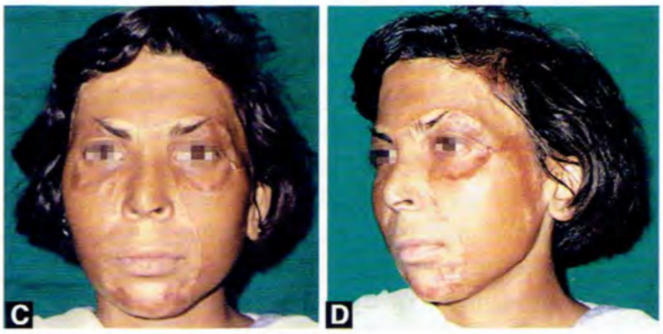


Fig. 34.3C: Appearance after spontaneous maturation of scars by physical therapy. The only operative intervention done is release and grafting of eyelids ectropion. Also note the spontaneous clearing of corneas after restoration of eyelid function



Figs 34.4A and B: Young girl with extensive post burn scars on whole of face



Figs 34.4C and D: Surgical correction by excision and cover with intermediate thickness split skin grafts from thigh on to cheeks, forehead and nose. Note good color and texture match on the face and excellent contour of the resurfaced nose. Patient opted for use of eyebrow pencil over surgical reconstruction of partial loss of eyebrows

34.29A to E). Last but not the least, they need psycho therapy to boost their morale. Most commonly the exposed areas like head and neck, upper limbs and feet are treated. It must be remembered that improvement should be seen in totality and not in isolation. It is of no use making the upper lip look like normal while the lower lip or forehead is 'ugly'.

The post burn scars may be atrophic, hypertrophic/ keloid, hypo/hyperpigmented and stable or unstable. The atrophic scars need intervention because they may breakdown with minimal trauma especially when present over areas of stress, e.g. joints, scalp, subcutaneous bones etc. and become unstable. When present over non-stress areas they are unesthetic. The atrophic scars need excision and cover with split skin grafts.

The deep dermal burn injuries and the donor sites of thick split skin grafts, both of which involve the reticular layer of dermis, usually heal by hypertrophic scar formation. These are hyperemic, red to deep purple in color, raised, gradually increasing in thickness, firm to hard in consistency and remain confined to the limits of the original burn injury. These lesions are itchy and some patients may even complain of pain or both. There is an over-abundant deposition of collagen during the proliferative phase of wound healing. The natural history of these scars is of spontaneous resolution with diminishing redness, thickness and itching over a period of 9-18 months when they are termed as mature. A number of factors can influence the severity and time period of maturation. Age of patient, anatomical site and depth of burn, presence of continued infection especially sinuses due to retained hair follicles, associated repeated tension due to joint movements are some of the important factors apart from individual genetic predisposition. The prevention and treatment of hypertrophic scars is discussed along with post burn contractures later.

A number of patients have the chief complaint of hypo or depigmented scars.[12] These vitiligo patches are very unsightly and are a taboo in our society. It may not be possible to treat all of them if they are extensive. However, in areas which cannot be covered by clothes like face, neck, hands and feet, treatment has to be done. Similarly, if limited areas are present over the breasts or anterior abdomen in females, treatment has to be offered (Figs 34.5A to 34.6B). The surgical treatment should begin after the maturation of scar and usually takes the form of excision/dermabrasion and over grafting with thin split skin grafts. Post-operative hyperpigmentation should be prevented by religious avoidance of exposure to sunlight. Although color may not be exactly like the surrounding skin but the results are always very satisfying when compared to initial problem. Very small areas may be treated by tattooing of appropriate pigment in the vitiligo area.

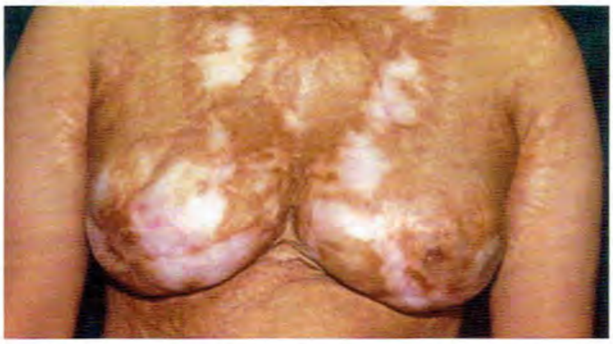


Fig. 34.5A: A 22-year-old female with hypopigmented post burn scars over both breasts

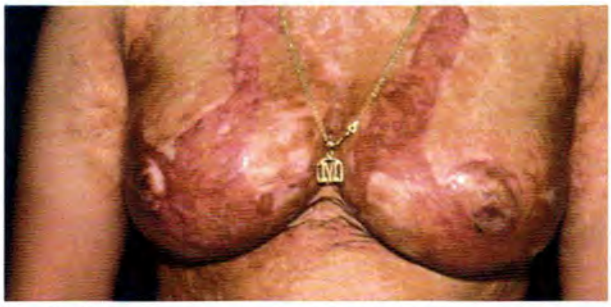


Fig. 34.5B: Early result after excision and over-grafting with thin split skin grafts

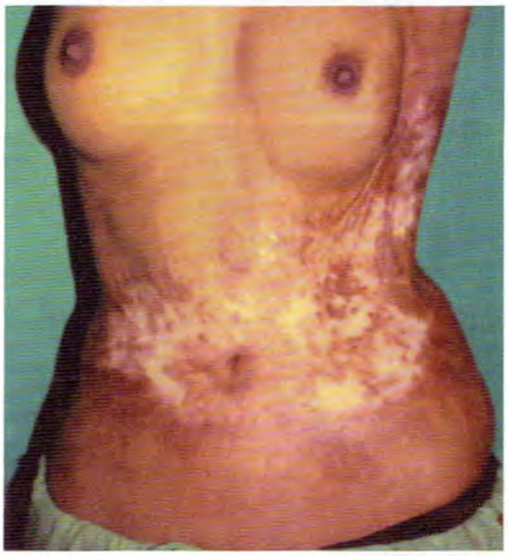


Fig. 34.6A: A 25-year-old female with hypopigmented post burn scars over abdomen, an area usually exposed while in traditional Indian dress "Saree"

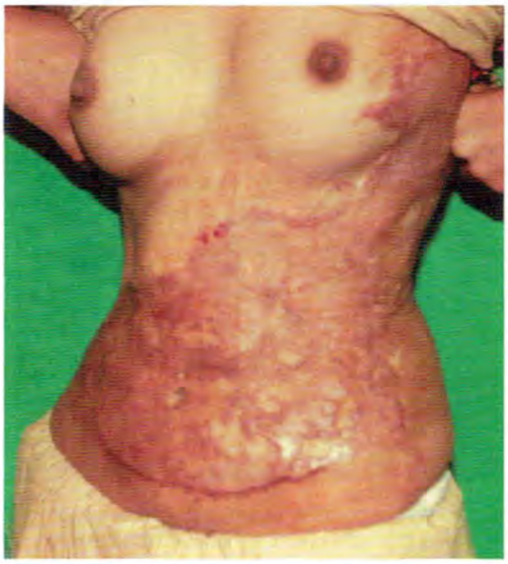


Fig. 34.6B: Result at 6 weeks after excision and over-grafting with thin split skin grafts

Patients may also present late with mature scars along with one or more of other post burn sequelae. They need detailed assessment of the functional and esthetic needs and planning of priorities and order of surgical interventions.

POST BURN CONTRACTURES[13,14]

A patient who receives the best of modern burn treatment is expected to heal without any contracture. However, contractures, like bed sores, do form even in the best of facilities. This is because contraction is a strong natural force and difficult to counter. Moreover, life-threatening complications in acute stage may compromise on problems which can be tackled later. Unfortunately, the incidence of post burn contractures is extremely high in our country. Quite often, they are not only multiple in a given patient but also very severe and diffuse. This is because the majority of the millions of burn victims in our country have no access to a trained burn specialist. The number of trained burn and plastic surgeons is too small in our country. It is around one thousand for more than a hundred crore (one billion) population. The burn patients are treated not only by a general surgeon but also by general practitioners of all systems of medicine, quacks (Fig. 34.7) and even patients and their relatives themselves. In this scenario, the aim and end point of a burn injury management is, unfortunately again a healed wound only. However, this healing (actually contraction) does not stop here and continues till multiple severe contractures result. A contracture is, thus, the end result of the process of contraction. When the contracture is opposite a joint, it leads to a functional deficit in its full range of motion. Thus, while contractures of the lower extremity interfere in upright ambulation and wearing of ordinary shoes, those of the upper extremity interfere in all the day-to-day activities of the individual. The non-joint contractures like ectropion of eyelids endangers vision and that of lips in normal eating and lip seal apart from poor esthetic appearance. It is at this stage that most of these patients move on to the metropolitan cities in search of a plastic surgeon who can now treat them. Patients of post burn contractures, defects and disfigurements constitute almost half of the workload of many general plastic surgeons, especially the ones in government institutions.

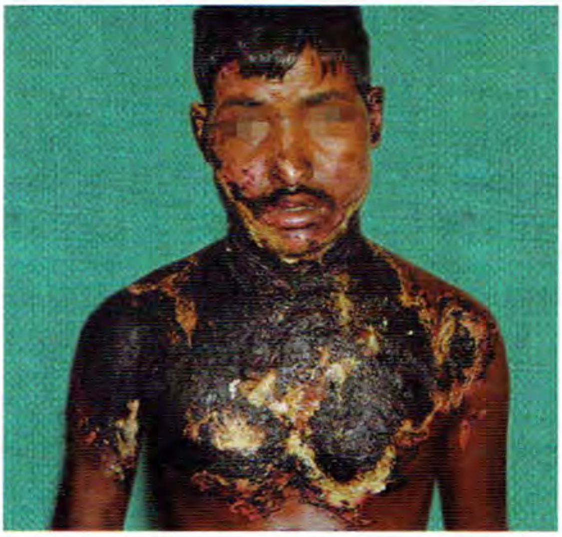


Fig. 34.7: Patient being treated by quacks with application of indigenous mixture of 'Ratanjyot' (an edible coloring agent) and camphor in coconut oil.

Mechanism of Burn Wound Healing and Development of Scar and Scar Contracture

An understanding of the burn wound healing[15,16] is fundamental not only to the management of the acute burn wound, but also for the prevention, minimization and treatment of post burn scars and scar contractures. The healing of a burn wound is accomplished in two ways, namely, complete regeneration (restitution) and substitution. Restitution is possible only if the skin is burnt as deep as the stratum papillare and all the specialized cells of the organ are preserved. The epithelial cells, in these cases, are derived from the epithelial appendages such as pilosebaceous units and sweat glands in the central portion and wound edges at the periphery. These appendages extend into the deeper dermis and may even penetrate into the subcutaneous fat (as in beard area in males) and survive in partial thickness injuries. The sequence of cellular events that comprise epithelialization include cellular detachment, migration, proliferation and differentiation. If the skin is affected deeper in the zone of stratum reticulare, the defect is covered by substitutive unspecialized connective tissue. The final result is demonstrated by a lesser or more extensive formation of the cicatrix. With full thickness loss of skin, wound contraction and epithelialization from the margins occurs leading to contractures.

Contraction is an active biological process by which an area of skin loss in an open wound is decreased due to concentric reduction in the size of the wound. It is a vital part of spontaneous healing of open wounds and perhaps nature's attempt for closure of the wounds. The reduction in size of wound causes lesser degree of connective tissue deposition and the amount of epithelialization needed is decreased. Wound contraction is a satisfactory mechanism when the tissue loss is small, in a non-critical area and surrounded by loose skin. A small full thickness burn over medial thigh, calf area or abdomen may heal satisfactorily by contraction aided by epithelialization from the margins. In a fingertip injury or small scalp loss, contraction may be desirable so as to have specialized sensitive and hairy skin respectively to cover the wound.

Scar contracture, on the other hand, is the end result of the process of contraction occurring in a healed scar. There is excessive scar formation across the joints resulting in a functional and esthetic deficit.

Associated Local Conditions in Patients with Contractures

A post burn contracture may be associated with additional problems on the skin and/or in the deeper tissues.

On the Skin

There may be:

i. A hypertrophic scar/keloid or an atrophic scar.
ii. An unstable scar over flexor aspects of joints/chronic non-healing ulcer(s) over non-joint areas which may or may not heal over prolonged periods of time/chronic postburn massive raw areas which are not expected to heal of their own ever (Figs 34.41A to 34.42B)
iii. Depigmented/hyperpigmented scar
iv. A Marjolin's ulcer.

Presence of one or more of above along with a contracture may alter the physical therapy and/or surgical treatment of a contracture. For example, an unstable scar or chronic non-healing ulcer(s) needs their surgical excision, release of contracture and provision of skin cover. Physical therapy in the form of exercises, massage and pressure garments prior to surgery is not possible in these cases. Bigger raw areas need wound closure with skin grafting before contracture can be subjected to physical therapy (Figs 34.41A to 34.42B) Wide excision of Marjolin's ulcer has to be combined with release of contracting bands (Figs 34.47A to E). A post burn contracture associated with a hypertrophic scar or an atrophic scar or a depigmented scar needs excision-release of all to achieve best results not only functionally, but also esthetically.

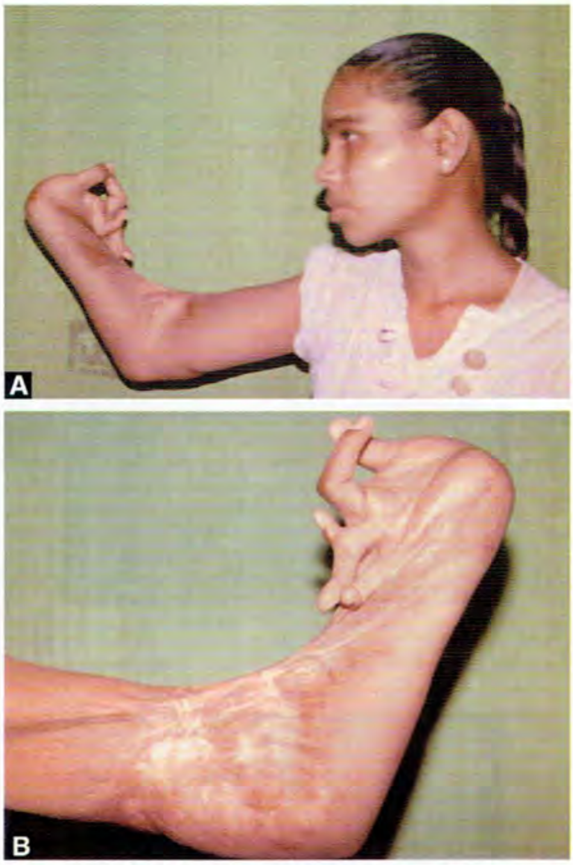
In the Deeper Tissues

The deeper tissues may be affected either due to their involvement in the initial burn injury (e.g. electrical burns) or secondary to the presence of a skin contracture over a prolonged period of many years which leads to shortening of musculo-tendinous units and neurovascular structures. Contractures of fingers following electrical burns are invariably associated with loss of underlying flexor tendons and need flap cover, even if a split skin graftable bed is present, because of the need for tendon grafting later. In addition, there is greater risk of digital circulation becoming compromised on straightening the finger as compared to patients with thermal burns. The joints may be subluxated or dislocated with joint capsule and ligaments becoming tight in the direction of the contracture especially when adult patients present for treatment of burns sustained in early childhood. The bones may be deformed specially in growing children, e.g. mandible may be deformed due to chronic pull in cases of severe post burn contractures of the neck (mentosternal contractures) (Figs 34.43A and B).

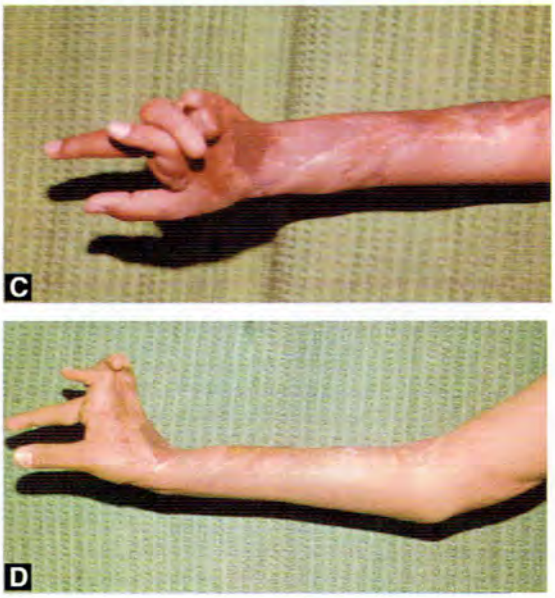
The shortening of deeper structures has great clinical significance. It may not be possible to release the contracture fully at the time of operation because the neurovascular structures, tendons and muscles may stand out as bowstrings limiting any further release. Moreover, the vessels may also go into spasm with compromise of distal limb or digit circulation due to excessive stretch. There may be prolonged neurapraxia due to nerve stretch. The muscles/tendons may resist any lengthening by forceful pull even under general anesthesia. Such contractures need release and dressing of the wound followed by sustained traction using various methods for complete correction. This can generally be achieved within two weeks. Skin cover is provided only after complete or maximum possible correction is achieved (Figs 34.12A to D). In occasional circumstances, when the patient presents with severe post burn contractures of elbow, wrist, knee etc. decade(s) after sustaining the burns in childhood, it may not be possible to achieve complete release and correction at all (Figs 34.8A to D). When bones and joints are also affected, orthopedic consultation may be needed. When mandible is deformed along with loss of normal dental occlusion in severe and untreated post burn contractures of neck in children, orthodontic/orthognathic measures are needed after the contracture is fully treated.

Timing of Surgery in Post Burn Contractures

As a rule, surgical intervention for post burn contractures should not be undertaken during the active phase of healing and scarring, i.e. as long as the scar is immature and highly vascular. This usually takes one year or so. One must allow the scar to become mature, soft and supple and 'avascular'



Figs 34.8A and B: Severe contracture of elbow and wrist secondary to burns sustained at the age of 1 year



Figs 34.8C and D: Suboptimal result after release and skin grafting is attributed to bony deformity and contracted ligaments, joint capsule and musculotendinous units

before undertaking surgery for contractures. This is because a highly vascular scar bleeds more during operation with difficulties in achieving perfect hemostasis resulting in poorer graft 'take' leading to healing by further contraction. Secondly, operation on an active, highly vascular scar with wound bed still in active phase of contraction, adds insult to already traumatized tissues with vigorous local response in the form of further contraction. All these lead to a less than perfect result. Moreover, an immature scar is amenable to physical therapy measures resulting in significant improvement even non-surgically. With the passage of time, some mild contractures may improve with a better final result than if they had been surgically managed. Hence, one must get benefit from this therapy before any operative intervention. Time is a great healer, not only of the emotional trauma but also of the wounds and scars of the skin! The patients also develop more realistic expectations during this waiting period, due to their interaction with other patients who are in their last stage of reconstructions. Lastly, it must be remembered that physical therapy is the mainstay of achieving best results even after surgical intervention. A patient who is non-compliant prior to operation is likely to get a poor or an average result postoperatively with all the blame being put on the surgeon especially in this era of Consumer Protection Act.

A burn surgeon must never be guided or influenced by the patient, his or her relatives, higher authorities or even politicians to do 'something operative' at the earliest. However, there are many exceptions to this general 'rule' of scar maturation. In these cases the results may be compromised and they may need another surgery at a later date.

i. Ectropion of the eyelids, especially the upper eyelid with constant danger of keratoconjunctivitis, corneal ulceration, scarring or perforation with loss of vision.
ii. Incapacitating contracture of the neck with inability to look forwards.
iii. Severe microstomia interfering with adequate nutrition and maintenance of orodental hygiene.
iv. Crippling contractures of hand,[17,18] especially dorsal contracture with metacarpophalangeal joints going in hyperextension leading to permanent damage to extensor mechanism with various deformities.
v. Contractures of both the knees, which force the patient to be on 'all the fours' and endangers the very dignity of being an upright human being.
vi. Post burn contractures with associated adjoining chronic raw areas needing skin cover.

vii. Contractures with infected hypertrophic scars and abscesses, which need excision/drainage for their recovery.

viii. Any severe, incapacitating contracture unlikely to improve at all with physical therapy measures.

Priority of Release in Cases of Multiple Contractures

A large percentage of patients coming to a burn surgeon have multiple contractures in different parts of the body. It is obvious that all of them cannot be released in one stage. In addition, there may be other reconstructive needs, e.g. reconstruction of scalp, nose, pinna, and face, etc. Quite often, there may be limitations due to paucity of healthy donor sites for skin grafts and flaps. Release of contractures and reconstruction in such patients may take many years. Therefore, a definitive strategy is essential in planning to reduce the number of anesthetic and surgical procedures and yet provide the best functional and cosmetic result safely. With each stage the patient must be able to appreciate the result and improvement. This adds to the surgeon's strength and patient's compliance and morale. It is ideal that this plan of action is put in black and white before the treatment is started and kept in patient's records along with a copy to patient.

An ectropion of the eyelids, especially the upper eyelid, with its attendant risks needs to be dealt with first to prevent irreparable damage. One must remember that loss of vision due to burns per se is rare expect for chemical burns (Figs 34.9A and B, 34.29A to E) It may even be managed under local anesthesia in case of an adult. Similarly, a severe contracture of the neck or microstomia leading to difficulties in intubation should be corrected before planning any other contracture release or reconstructive procedure requiring general anesthesia. This takes care of repeated intubation difficulty with its all the attendant risks. Additionally, the complete release of a neck contracture removes the extrinsic pull on facial structures (lips, chin and even lower eyelids) and contractures of the axilla and the breast. For full release, the neck contractures need division of platysma and fascia until the strap muscles are seen.

Contractures of the hand need to be addressed after the neck is treated. They are not only crippling and interfere in the day-to-day life, studies and earning, but may also cause permanent damage to the delicate balance of tendon movement. This is especially true of contractures on the dorsum of hand. A dorsal contracture of the hand with metacarpophalangeal joints in extension not only stops the function, but also wrecks the machinery (extensor apparatus) (Figs 34.10A to D). Here, it must be noted that both hands should rarely be treated together (except in small children) to allow the patient his/her daily needs of feeding and toilet care (Figs 34.11A to D). Also, the hand with greater

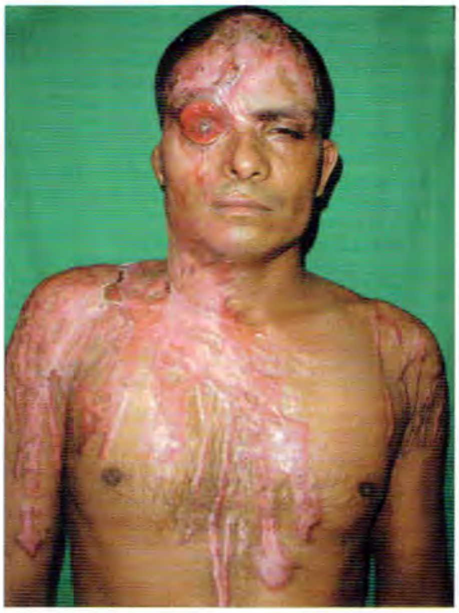


Fig. 34.9A: Young adult with very severe ectropion of right upper and lower eyelids, loss of vision, with facial disfigurement

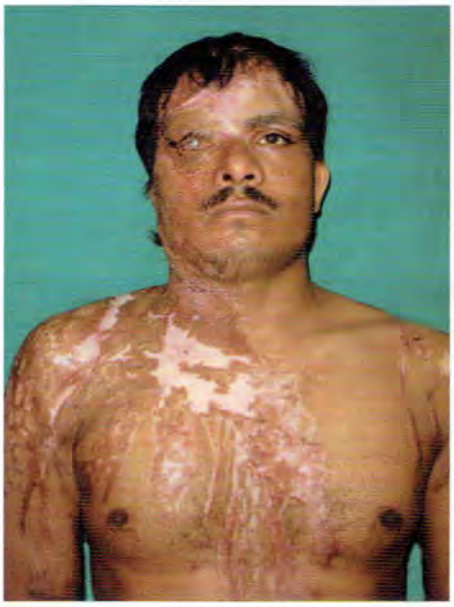


Fig. 34.9B: Simultaneous release and split skin graft of both eyelids done without waiting for scar maturation. Result after 4 months. Please note the maturation of other scars with massage and passage of time

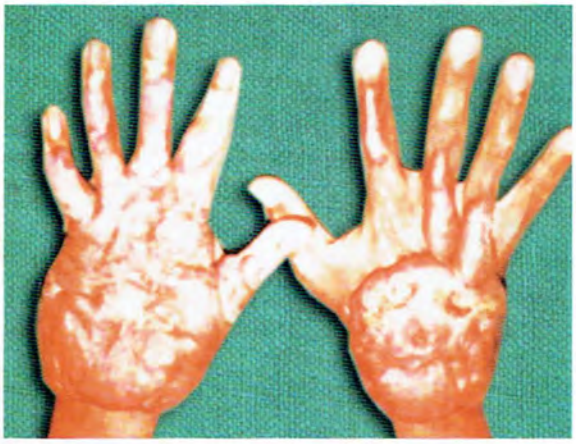


Fig. 34.10A: Post burn contracture and severe hypertrophic scar on dorsum of both hands and wrist

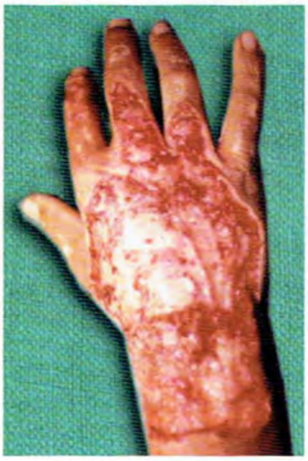
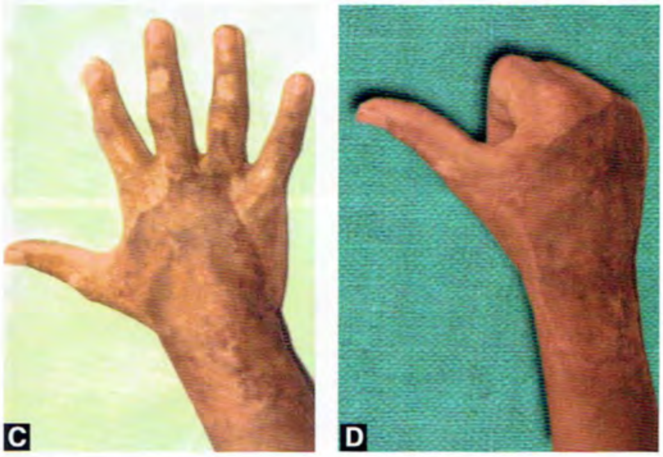


Fig. 34.10B: Intraoperative: Complete excision of hypertrophic scar and release of contracture (Right hand)



Figs 34.10C and D: Result at 1 year with full range of movement at MP joint with pleasing esthetics

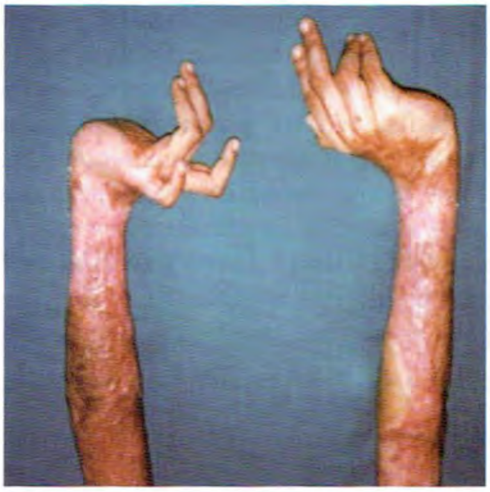
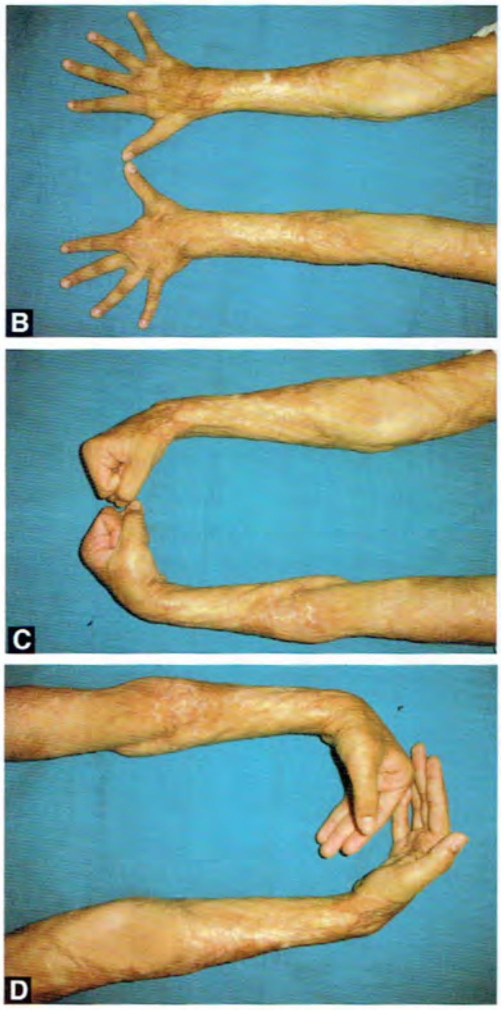


Fig. 34.11A: A Post burn contracture on dorsum of both hands and wrist in an 8-year-old-child



Figs 34.11B to D: Release and split skin grafting with complete restoration of hand and wrist function. Result after 5 years

likelihood of becoming more functional should be treated first rather than the right hand (in a right handed person), which may be almost irreparably damaged. Conversely, the hand which may be retrievable today and considerable damage is expected with any further delay, should be operated first, even if severely affected. When axilla, elbow and hand are all affected on one side it may not be possible to operate on the hand till the axilla and elbow are released.

Contractures of the popliteal fossa also need early intervention as they are also highly incapacitating (Figs 34.12A to E).

The order of management of other contractures depends upon the surgeon's evaluation of the remaining reconstructive needs, feasibility of combining multiple procedures in the same surgical sitting, availability of donor sites, scope of improvement and the patient's desires.

Prevention of Wound/Scar Contraction and Scar Contractures

In a healed burn patient, the healing may have been achieved by one or more of the following:

i. Spontaneous healing with epithelialization and contraction.
ii. Split-skin grafting over granulating raw areas after spontaneous eschar separation
iii. Early excision and split-skin grafting.

In all these patients, various physical therapy measures are immediately instituted for the healed burnt areas and split-skin donor sites to minimize scars and scar contractures. It is to be noted that physical therapy in a burn patient actually begins on the day of admission. This may take the form of limb elevation, crepe bandages to reduce edema, active and passive joint movement, splintage and positioning of the body parts in anti-deformity posture, functional position, etc.

Due to prolonged contractile nature of the burn wound, a patient who heals initially with full range of motion may develop severe deformities as the healed skin/scars mature and hypertrophic scarring occurs. Dermatological problems, e.g. blister formation, its breakdown, excessive dryness and cracking can occur or a gradual build up of hypertrophic scar tissue can lead to a rapid decrease in motion and functional ability. Thus, the physical therapy measures have to be continued till the scars have matured and no further change is expected. During continuation of this therapy, various surgical interventions may also be needed and undertaken. It is only when all the scars have matured, the

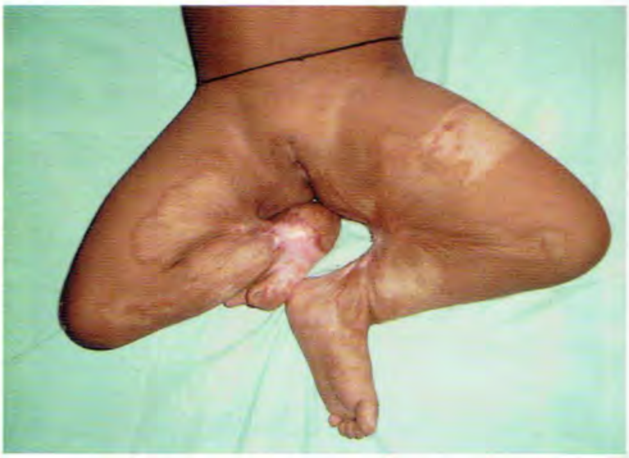


Fig. 34.12A: Severe contracture of both knees and right ankle in a 3-year-old girl precluding ambulation

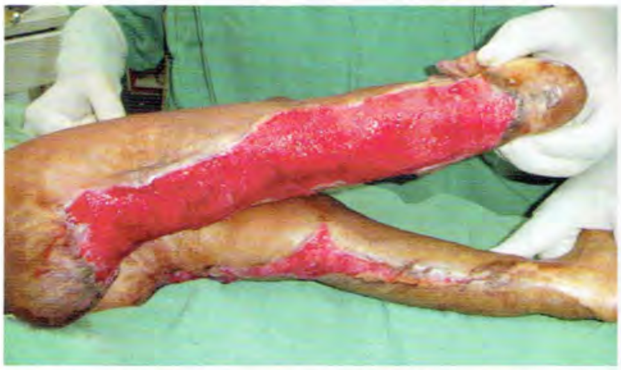
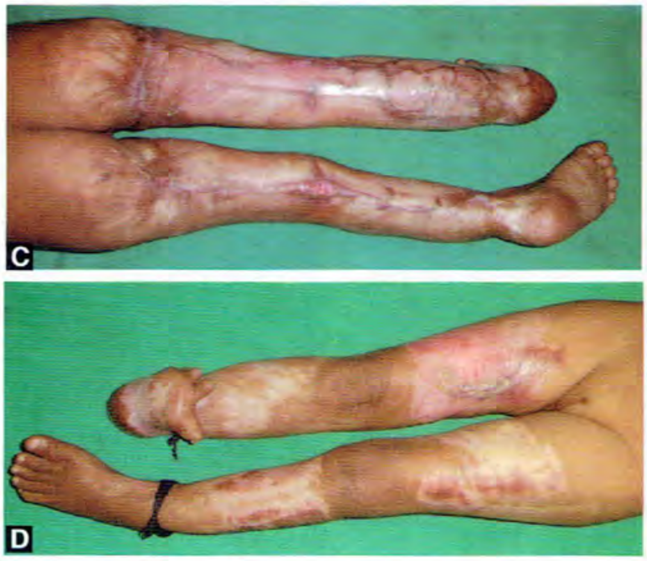


Fig. 34.12B: Complete release was not possible in one go; serial splintage done over 2 weeks. Please note the straight limbs and healthy granulations



Figs 34.12C and D: Grafted and all wounds healed

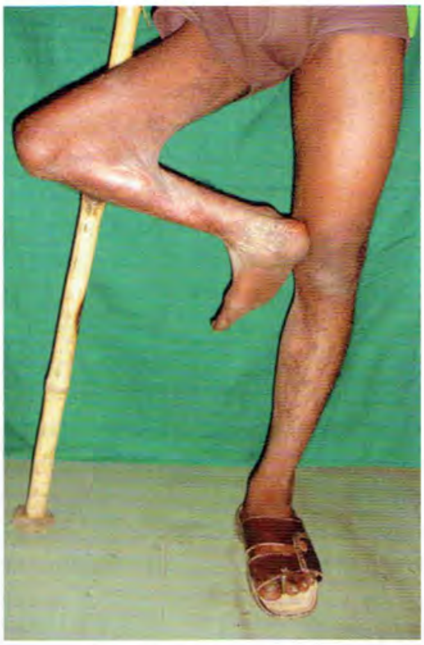


Fig. 34.I2E: An untreated popliteal and ankle contracture due to burns sustained in infancy leading to limb shortening with subluxated knee joint

correctable deformities looked after, reconstructive surgeries performed and no further reasonable improvement is expected that the treatment of a burn patient is called off (except probably continued psychological support and aid for return to society).

The control of wound/scar contraction and scar contracture is done by the following techniques:

1. Skin Grafting[19-22]

The single most important and effective method of controlling the wound contraction is to close the wound at the earliest. This is done using split-skin grafts in deep dermal and full thickness burns. Contraction can be inhibited by applying grafts to fresh wounds (as in early excision) or over healthy granulating areas (after eschar separation). Full thickness skin grafts inhibit contraction almost completely. Unfortunately, in a clinical setting, the raw areas of burns have to be covered with split-skin grafts so that the donor sites can heal on their own and may even be re-harvested. The split skin grafts may have to be expanded with meshing. Although this leads to complete healing of the wound, the latter is largely covered with epithelium in the interstices of the meshed graft.

It is widely believed that thicker the graft, greater will be the inhibition of contraction. This holds good only if the grafts are harvested from a given site. The fact is that when split-skin grafts are harvested from different sites, it is the total percentage of dermal thickness grafted which determines how much contraction will be inhibited. Delayed application of skin graft does not inhibit contraction as effectively as immediate grafting.

2. Pressure Garments[23-27]

Deep dermal burns and donor sites of intermediate and thick split-skin grafts frequently develop hypertrophic scars. They must be prevented or treated using pressure garments (Figs 34.13A to F). These compression garments quickly reduce the mass of immature hypertrophic scars. The effectiveness of these garments decreases after the development of mature

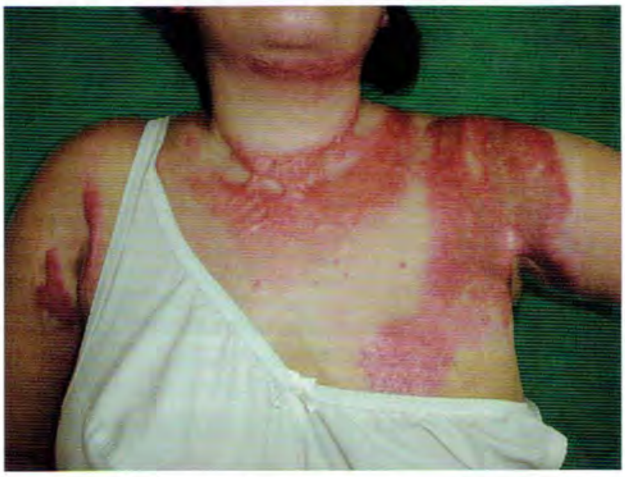


Fig. 34.13A: Post burn hypertrophic scar in healed deep dermal burns

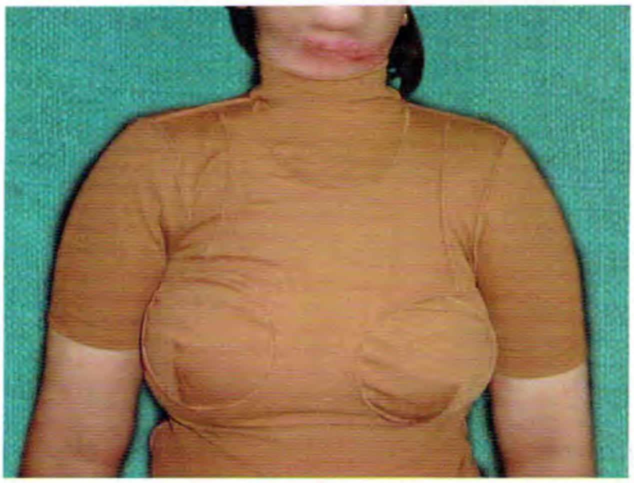


Fig. 34.13B: Pressure garment

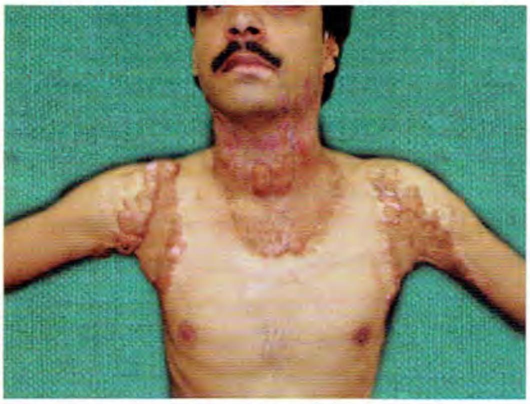


Fig. 34.13C: Post burn hypertrophic scar in healed burns

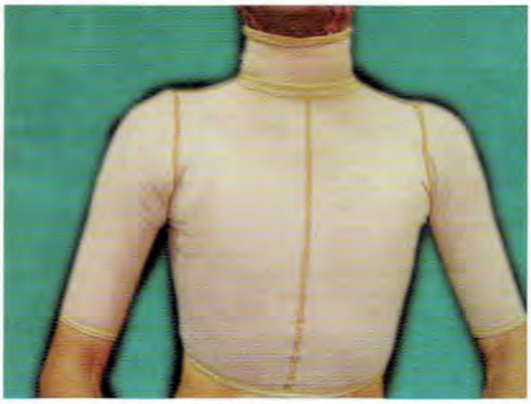
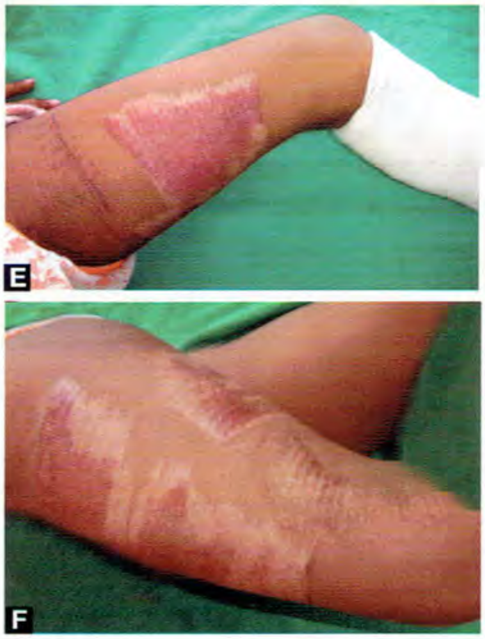


Fig. 34.13D: Pressure garment



Figs 34.13E and F: Hypertrophic split skin graft donor site being treated with pressure garment. Note the markings of the garment cloth on the skin on turning down the pressure garment

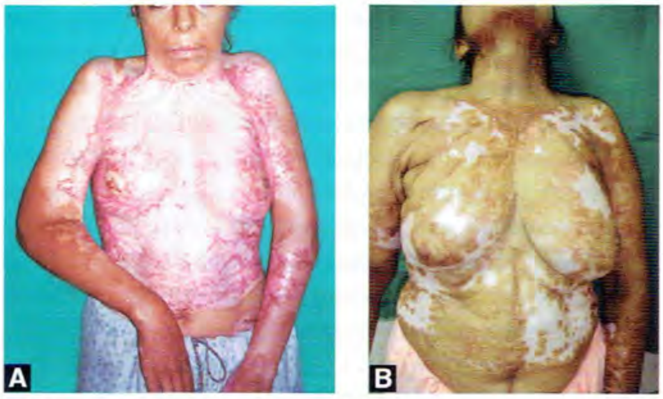
hypertrophic scars. Before prescribing commercial pressure garments, the newly healed skin must be preconditioned to accept the stress and pressure exerted by the garments. For this, initially gentle pressure is applied with crepe bandages. As the skin 'toughens', the commercial pressure garments may be prescribed. In the hot and humid conditions of our country, it is difficult to wear them continuously for a period of 24 hours a day. Hence, compliance is a great problem not only in children but also in adults. They have to be worn for a period of at least 9-12 months to be of benefit.

Although application of pressure to influence the abnormal scars has been used for the last two centuries, it is only in the last fifty years that it has been popularized and its mechanisms of action studied. A reduction in fibroblast content, total chondroitin-4-sulphate, cohesiveness of collagen fibers and rapid disappearance of collagen nodules (normally found in hypertrophic scars) and reorientation of the collagen bundles have been noted. A reduction in intralesional mast cells and their histamine production and release has also been demonstrated. Pressure therapy diminishes the number of myofibroblasts, erythema, thickness and firmness of hypertrophic scar and accelerates its maturation.

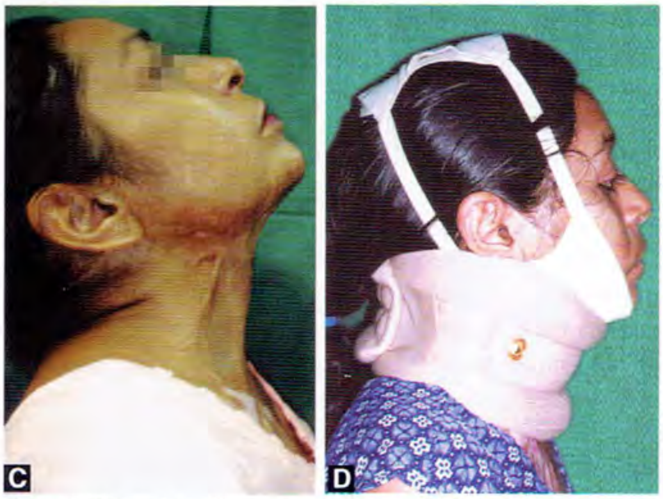
The exact mechanism(s) whereby the pressure therapy works is still not known. However, the various explanations put forward for these changes are (a) hypoxia of the scar tissue by occlusion of the microvasculature, (b) increased collagenase mediated collagen breakdown due to pressure induced decrease in capillary blood flow, (c) reduction in tissue edema with less ground substance due to compression (d) neutralization of local venous hypertension by preventing leakage of plasma proteins and improving tissue oxygenation (e) elevation in skin temperature by 1-3degrees by blockage of heat loss from the cutaneous surface, etc. The ideal pressure required is also not clear but it must be more than 24 mm Hg for capillary pressure to be countered. While significantly lower pressures may not work, a pressure above 40 mm Hg may not be tolerated with appearance of paresthesias and maceration.

3. Splintage and Exercises[28,29]

The scar collagen and elastin are relatively uncross-linked and malleable during their initial deposition. Gentle, passive and sustained stretching using various dynamic and static splints exploits this malleability and is an effective technique for the lengthening of bands of scar tissue and increasing the range of motion (Figs 34.14A to E). Combination of paraffin wax bath application with positions of sustained stretch using serial splintage is highly effective. When several



Figs 34.14A and B: (A) A 27-year-old female with multiple contractures and immature, hyperemic scars. (B) Result 18 months later, the contractures have been treated and the scars matured with greatly diminished vascularity. Please note the improved breast contours due to diminished upward pull and softening of mature scars. No surgical intervention can be worthwhile for extensive hypopigmented areas



Figs 34.14C and D: (C) Postoperative lateral view of neck. (D) Chin-neck pressure garment and cervical collar is worn for 6-9 months to provide splintage for favorable maturation of graft

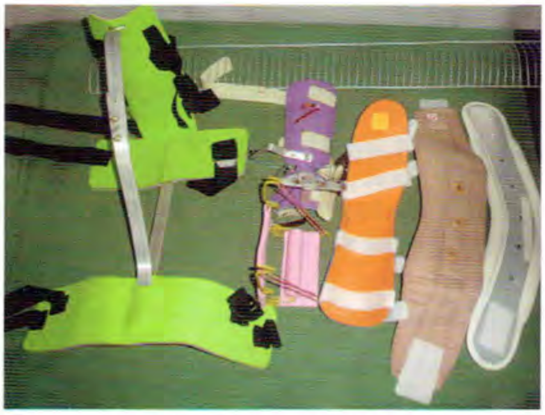


Fig. 34.14E: Various splints used for post burn patients

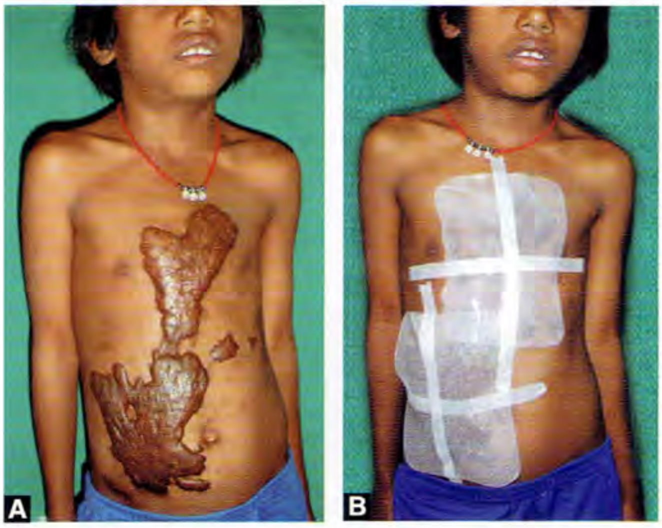
joints are involved, the contractile nature of the scar must be remembered and body positions devised that provide stretch along the length and breadth of the area involved. Thus, in patients with scars involving both of the upper limbs, axillae, anterior chest and neck, a pillow is placed under the shoulder and upper limbs are kept in maximum abduction. The paraffin is applied to the patient in this position and left in place for 20-30 minutes of sustained stretch.

4. Massage

The healed burn wounds and donor sites of split-skin grafts are massaged with lubricants like coconut oil or one of the various proprietary creams and oils. The massage causes increased flow in the veins and the lymphatics thereby increasing the absorption of metabolic wastes. There is an increase in capillary circulation and mobility of subcutaneous soft tissues. The lubricants are applied lightly and rubbed in gently after a thorough bath. Heavy lubrication is avoided as their build-up on the skin causes pimples and furuncles. Massage should be done with care to avoid friction to superficial layers of skin (neoepidermis/scar epithelium) that can cause blisters. This is due to the absence of desmosomes between neoepithelium and underlying tissues. There are psychological benefits also as it helps to relax the patient and creates a feeling of well-being. Last but not the least, it buys time for the surgeon before the time is appropriate for surgical intervention.

5. Silicones[30]

The silicones have been used in various forms for prevention and treatment of hypertrophic scars (Figs 34.15A and B). These are synthetic polymers based on dimethylsiloxane monomer with polydimethylsiloxane (PDMS) being the commonest in use. Silicone gels having lightly cross-linked chains of PDMS are available as sheets and have to be worn for 24 hours a day with particular care being taken of local hygiene to avoid the development of contact dermatitis and folliculitis. The exact mechanism of action of silicone gel sheets is not known. It may exert its effects by increasing the temperature of the scar, thereby enhancing the activity of collagenase, which is known to increase several folds over 1 to 2°F increase of body temperature. Other effects of silicone gel, such as increased pressure, lowered oxygen tension and occlusion may be less important. Hydration of the stratum corneum and direct release of low molecular weight silicone fluid into the scar are other possible modes of action. However, silicone does not appear to enter scar tissues. The biggest drawback of silicone gel sheets is the practical difficulty in keeping the sheet in contact with the scar all the day and night and their application on exposed areas of the body like face is unsightly.



Figs 34.15A and B: (A) Post burn hypertrophic scars over anterior trunk (B) Application of silicon gel sheet

Silicone in the form of gels, creams and ointments containing short, straight and unbound chains of PDMS are being increasingly promoted as they are simple to use, do not need any tapes to hold them, are not unsightly and maintaining hygiene is easy. A very thin film is applied over the hypertrophic scar and allowed to dry over 4-5 minutes. On drying, it forms a thin, impervious and almost invisible film. It is applied twice daily. Xeragel®, Vaniza gel®, Kelo-Cote® etc. have recently become available in the Indian market.

6. Intralesional Steroids

Intralesional injections of triamcinolone, every 4-6 weeks, have been in use for over 40 years and found to be effective for control of hypertrophic scars and keloids. Triamcinolone is a powerful steroid that exerts strong anti-inflammatory activity. It can be used in early stages of hypertrophic scar formation when the scar is comparatively soft and easy to inject. It can also be used in mature scars but because of their hardness it may be very difficult to inject especially in first couple of sittings. On being injected into a hypertrophic scar, it activates the endogenous collagenase to degrade the mature collagen to more soluble forms. After a number of injections the hypertrophic scars become flattened, softened and non-pruritic. However, the dose and amount injected at any one time is limited for a very small lesion only. Not more than 80-120 mg should be injected in adults in one sitting in a concentration of 8-10 mg/ml. The available vial of 40 mg/ml. can be diluted with 3ml of 1% lignocaine to decrease the pain of injection. Addition of hyaluronidase may improve the spread of drug into scar tissues. The main complication of atrophy is due to over-dosage and spillage into normal skin or subcutaneous tissues. Telangiectasia and hypopigmentation can also occur. Hirsutism can be an annoying problem in young females.

7. Management of Pruritis[31,32]

Itching is an extremely common complaint of patients with healing and healed burn wounds and donor sites especially at night. Patient's scratching the area to get relief causes breakdown of recently formed neoepithelium with consequent ulceration, the healing of which again causes itching, thus forming a vicious cycle. The ulceration(s) may become infected. This prevents application of many of the above-mentioned measures for minimizing scar contraction and optimizing its maturation. Even after many years, the patients may still complain of this problem. Pruritis affects the daily life and emotional well-being reminding the traumatic event again and again. In some patients this is perceived as pain although the mechanism of both is same. The treatment of post burn pruritis is difficult and frustrating to the patient and the burn surgeon alike. Oral antihistaminic drugs like pheniramine, promethazine, cetrizine etc. are commonly prescribed, especially at bedtime. Steroid containing creams, if at all used for local massaging, must be avoided over areas exposed to sunlight for fear of hyperpigmentation. As the dry skin is especially prone to itching and the healed areas may be deficient in production of sweat and sebum, keeping the skin well lubricated with moisturizers and oils is very essential. Newer therapies in the form of Gabapentin, H1 and H2 blocker combination therapy, topical application of histamine receptor blockers and transcutaneous electrical nerve stimulation have been introduced with variable results.

Surgical Intervention

The surgical management of any post burn contracture involves the following steps:

Release of Contracture

Complete release of contracture should be done avoiding damage to any important underlying structure, e.g. arteries, nerves, tendons, etc. Although contraction occurs in all directions, the incision should begin across the point of maximum tension, i.e. where the contracture is most tight. This point is usually opposite the joint line. The incision is deepened all the way to the unscarred, healthy tissues. After the complete release of contracture, multiple darts are made at appropriate points along the periphery of the defect created to take into account the contracture along other directions. Fish- tailing of the incision line at either end is inadequate and inferior to the multiple darts especially when the former are marked on the skin at the beginning of operation. Darts are 'cut as you go' while the fish-tails are predetermined. No attempt should ever be made at undermining the surrounding healthy/scarred skin and advancing it to decrease the defect. Incision line can be infiltrated with 1:200,000 adrenaline solution either alone or preferably mixed with lignocaine one percent to have a relatively bloodless field (Maximum dose of adrenaline is 5 micrograms/kg body weight and that of lignocaine is 7 mg/kg body weight). The limb contractures, especially distal to arm/thigh, should be released under tourniquet control, whereever possible. Bipolar cautery should be used to achieve accurate hemostasis during release incision. When a contracture is released under tourniquet, hemostasis is done once again after deflation of tourniquet. The raw area should now be covered with1:200,000 adrenaline solution soaks and skin graft harvested.

Incision versus Excision

In general, a contracture should be released by incision rather than by excision. This is especially true for patients who have received adequate preoperative physical therapy and their scars have become soft and pliable. Incision alone decreases the requirement for skin cover. When the scars are extensive, it is futile to excise a small amount if whole of scar cannot be excised for fear of creating an extensive raw area. Excision may, however, be required in certain circumstances e.g., (a) small adjoining depigmented or hypertrophic areas, excision of which will add to the final aesthetic result (b) atrophic/unstable scars/chronic non-healing ulcer(s) should be excised along with release of contracture to obtain healthy bed for split-skin graft 'take' (c) scars may also be excised so as to apply the graft/flap in accordance with principles of esthetic units. Partial excision of hypertrophic scars may sometimes be done, e.g., in a case of post burn contracture of neck, the scars may extend from chin, neck on to the chest and even abdomen. Here, only neck scars are excised. Before excision-release is planned, the size of the defect created must be estimated beforehand and availability of required donor site for skin graft/flap assessed.

Immediate versus Gradual Release

In general, a contracture should be released completely on the table in one go. However, in severe long-standing contractures, there is considerable shortening of musculotendinous units and neurovascular structures. Hence, it may not be possible to achieve complete release. Their exposure may limit any further release and vessels may go into spasm if forceful extension is attempted, especially in contractures of limbs. Similarly, when the joints are subluxated or dislocated, immediate complete release may not be possible. In all these cases, contracture is released as much possible and the wound is dressed. Full correction is achieved gradually over a period of up to a couple of weeks using serial splintage with Crammer wires, skin/skeletal traction or the modern distractor systems. Once full/maximum correction is achieved, skin cover is provided (Figs 34.12A to D). If split skin graft is applied immediately, the smaller size of the graft applied limits the full extension and/or else a halo of raw area appears around the graft on attempted serial splintage. The graft 'take' on uneven raw area may also be less than satisfactory. In addition, any future attempt at release may not be possible because these structures will be lying just under the graft.

Provision of Skin Cover

After the complete release of a post burn contracture, the recreated defect is covered using skin grafts or a skin flap. Commonly, the raw areas resulting after release of post burn contractures are covered with split skin grafts. Flap covers are used in special situations only.

Skin Graft: When using split skin grafts, sheet grafts are to be preferred and no attempts should be made to expand the graft by meshing. As far as possible, try to feed in graft more than the size of the defect to take care of the post-operative, inevitable secondary contraction. The junction line of the sheets of the grafts should preferably lie parallel to the axis of joint motion. Ordinarily, the skin grafts are applied immediately after complete release. Sometimes, delayed application after 5 days, once the granulations form, is done. The delayed approach is required when release of contracture results in exposure of fat which is a poor bed for graft 'take'. When the raw areas are oozing a lot, or when the contracture is associated with infected areas, delayed application gives a better result. When skin grafts have been used, rigid immobilization is done by one or more of the various techniques, viz. 'tie-over' dressings, plaster of Paris splints, crepe bandages, elastoplasts, etc. depending upon the site.

Split Skin Graft versus Full Thickness Skin Graft

In general, all contractures should be treated with split-skin grafts of intermediate or thick variety. This allows the donor site to heal spontaneously. Moreover, extensively burnt patients with multiple contractures have paucity of donor sites. Full thickness grafts, although better than the split-skin grafts in most of the properties, have poorer chances of 'take' and their donor sites in turn need split-skin grafts. Their use is limited to very small defects resulting after release of contracture in esthetically important areas, e.g. ectropion of the upper lip, lower eyelid, etc.

Skin Flaps: There are a few situations where a skin flap is a must. If the contracture release is likely to open up the joint, especially of the hands and feet, or tendon/nerve surgery is planned at a later date, e.g., old healed electrical burns, a flap cover must be provided after release of contracture. If release of contracture results in a moderate defect in a cosmetic area, which if covered with a flap will give a better aesthetic appearance, a flap cover may be provided. For example, an ectropion of the upper lip in an adult male can be released and covered with a flap from the scalp or upper neck to restore moustaches. In a female, the same ectropion requires a graft cover. A groin contracture may be treated using a tensor fascia lata flap with graft on the flap donor site (Figs 34.16A and B). This decreases the need for postoperative splintage to prevent secondary graft contraction.

If a local flap in the form of Z plasty, V-Y plasty, V-M plasty, etc. (for linear/webbed contractures) will be sufficient in itself to treat the contracture, there is no reason why they should not be used alone (Figs 34.17A and B). However, these flaps are always in danger of necrosis when raised in scar tissues. Moreover, they are possible only in very mild cases. They are also useful when massive diffuse contractures are treated with split skin graft and later on, after graft maturation, contracting bands form at the graft-surrounding skin junction. The flaps used for provision of cover may be local, regional or distant. They may be free or pedicled.

The biggest advantage of using a flap for treating a post burn contracture is that post-operative prolonged splintage

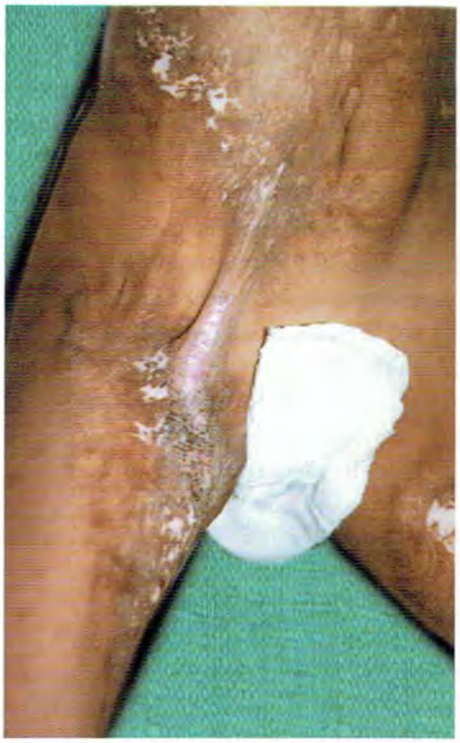


Fig. 34.16A: Young adult female with contracture of right groin region with umbilicus pulled downwards and laterally

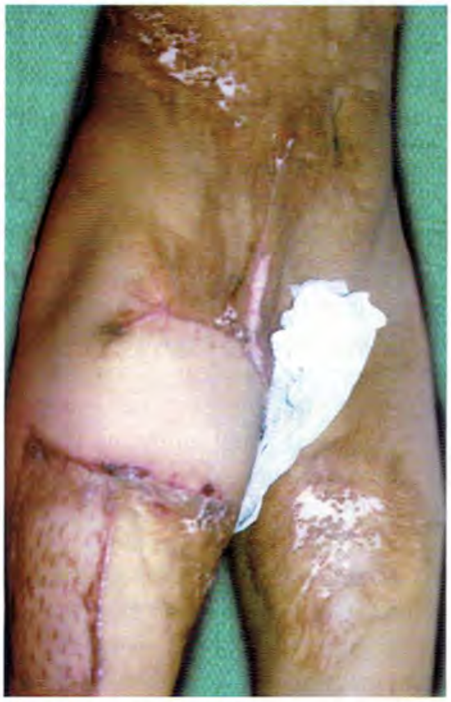


Fig. 34.16B: Release and cover with transposition of tensor fascia lata flap whose donor site was split skin grafted. Postoperatively the patient does not require any splintage

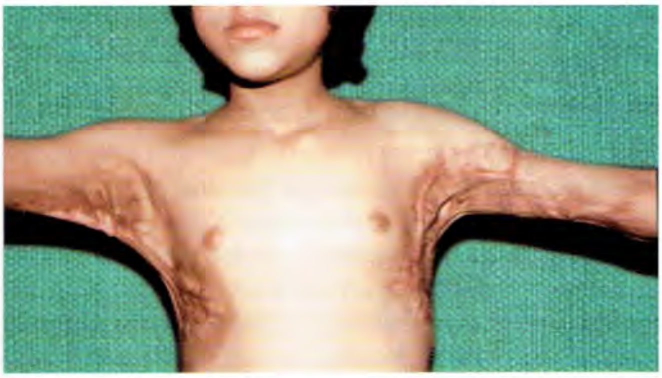


Fig. 34.17A: Contracture of both axillae

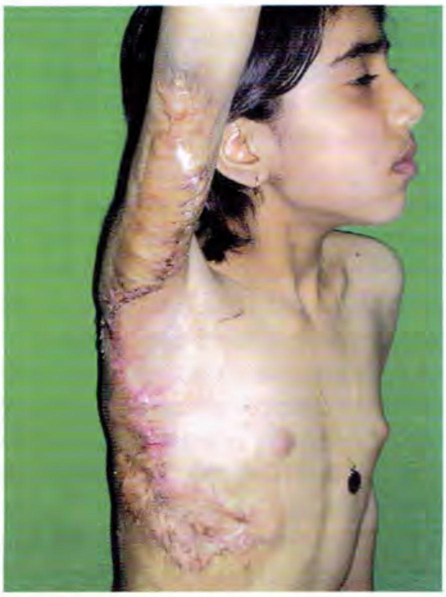


Fig. 34.17B: Right axilla treated by release—excision of scar contracture and multiple Z plasty

is not required. However, in a practical clinical scenario with patients of extensive burns with multiple sequelae, the appropriate flap may not be available. Moreover, the healthy unscarred skin of the flap may stand out prominently in the midst of surrounding scarred or skin grafted areas. Their donor sites also need split skin grafts for closure. The grafts blend far better in such surroundings. In patients with smaller burns and limited needs for reconstructive surgery, an appropriate local, regional or distant flap may be used to provide cover after release of contracture.

Donor Sites. The split-skin grafts are usually harvested from thighs. However, in a severely burnt patient, with extensive scarring, the grafts may have to be harvested from legs, upper limbs and abdomen, back or even scalp. While grafts from the limbs can be harvested using Watson's skin grafting knife, electrical dermatome is ideal for abdomen and back. It should be noted that in every patient who presents with multiple post burn problems which need to be addressed surgically, one must look for the donor sites available. In a case with multiple, massive contractures, the donor sites should be checked and a plan charted out for 'which donor site for which contracture'. This is important as large sheets are usually required for neck, axilla and facial resurfacing, etc. while comparatively smaller pieces of grafts are adequate for contractures of fingers, eyelids, etc.

Postoperative Care

Maintenance of released/corrected position is mandatory until the graft has become stable (usually 3 weeks) or till the flap margins have healed.

Postoperative use of static or dynamic splints, interspersed with a routine of daily physical therapeutic exercises is required to keep the joints in full range of motion (especially if static splintage is used). This therapy is continued till the grafts have matured (usually 9 to 12 months) and complete range of motion is achieved.

Maturation of the graft is aided using massage with lubricants, use of pressure garments and silicone gel sheets, etc. for a period of 1 year or more, till the graft loses its tendency to contract and can be pinched and mooved over the recipient area. Lubrication of the split skin grafts over large areas should be advised indefinitely because of absence of sebaceous and sweat glands in the grafted skin.

The detailed account of surgical technique for the correction of each and every individual post burn contracture-deformity and various local, regional and distant flaps described in literature for providing cover is beyond the scope of this chapter. The commonly seen contractures are ectropion of the upper and lower eyelids and lips, neck, axilla, elbow, hand, breast, groin, knees, ankle and toes. From time-to-time, many workers have tried to classify the contractures of different sites. The present author does not find them useful in treating his patients. Classifying them as mild, moderate and severe is of no consequence as a mild contracture of lower eyelid causes epiphora, upper eyelid may endanger cornea and popliteal fossa leads to an unstable scar-ulcer and even development of malignancy. On the other hand, a moderate flexion contracture of fingers or axilla may allow normal daily functions to be carried out. Moreover, it is difficult to quantify what constitutes mild, moderate or severe for different areas and in different individuals. Similarly, even classifying on the basis whether the contracture is linear or diffuse does not always mean that the former can be treated by a local Z plasty or similar other technique. If the surrounding area is scarred and/or transverse laxity is unavailable, raising any flaps may not be possible or else it may lead to their necrosis.

Ectropion of the Eyelids

As discussed above, this is the most important contracture which should not wait for scar maturation for its correction. It does not even need the patient being fit for general anesthesia as it can be very easily done under infiltration locally with xylocaine and adrenaline solution in adult patients. This infiltration is required even if the operation is being done under general anesthesia as the tissues are very vascular. The amount of split skin graft required is also very small and can be harvested under local analgesic infiltration or even simple application of EMLA cream (a eutectic mixture of 2.5% lignocaine and 2.5% prilocaine applied as a thick layer over the donor area for 2 hours before harvesting). An incision is made 2-3 mm away from the eyelash margin. With the help of gentle traction on the stay sutures applied at the lid margin, the contracting bands are gently incised with the slight touch of No. 15 blade and the ectropion released and over corrected. An intermediate thickness split skin graft is harvested and grafted with tie-over sutures. If both the eyelids have ectropion, both are released with over-correction and then grafted (Figs 34.3A to C, 34.9A and B) The conjunctival sac is washed with saline, an antibiotic ointment instilled into it and dressing done. Over-correction is very important as the postoperative graft and scar contraction can not be prevented due to free margin of the eyelids. When this operation is carried out in acute stage as is generally the case, re-ectropion although less severe is possible. However, the cornea gets protected and the second definitive operation can be delayed. In cases where the scar is mature at the time of operation, full thickness graft can be used for lower eyelid and thick split skin graft can be applied after upper eyelid release. One eye should be operated at a time as operating on both the eyes together leads to temporary blindness due to dressings for a period of a week or so. This is highly distressing and frightening to any person with otherwise normal vision. However, if there is vision in only one eye, both eyes can be treated together. It is traditionally believed that the ipsilateral upper and lower eyelids should not be treated in a single stage because of inability to achieve the required over-correction in these cases. But, the author's experience of over two decades is contrary to that. A V-Y plasty can be done only for lower eyelids when the eyelid skin is soft and supple and correction required is only 5-6 mm.

Mentosternal or Flexion Contractures of the Neck

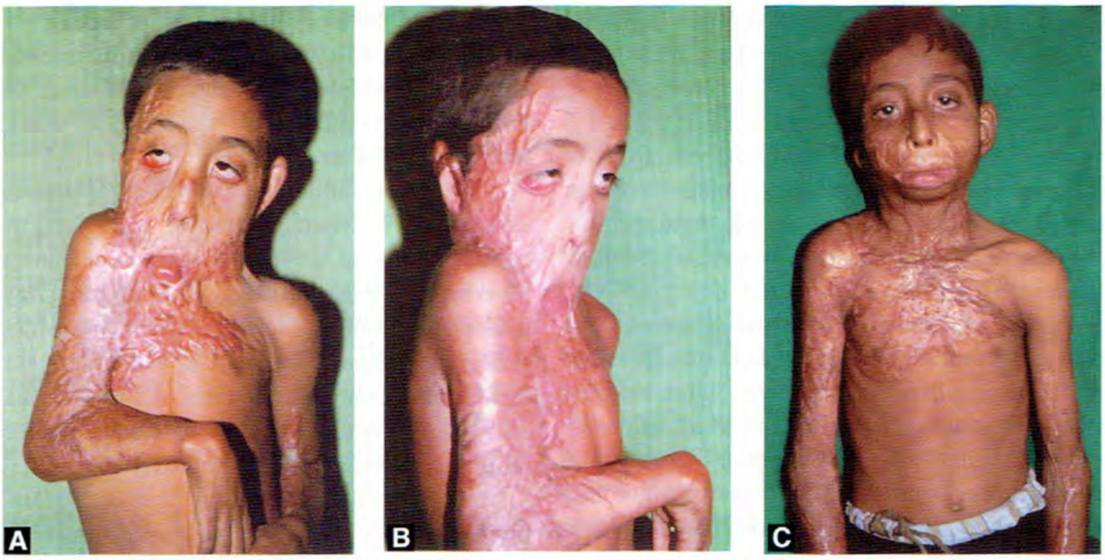
These are very common because the skin of the anterior neck is very loose and patients tend to use pillows under their head either unknowingly or even otherwise. These may become combined with ectropion of the lower lip and may be severe enough as to bring the vermilion border of lower lip and chin to the level of manubrium sterni. Very commonly they are diffuse and may extend to mid-lateral line on either side. They may involve skin of one half of the neck so that the neck is flexed to one side or else there may be linear band(s) as may occur after chemical burns. All severities of post burn neck contractures are seen from very mild cases with almost no disability to the severest ones who have microstomia, deformed mandible and poor occlusion and kypho-scoliotic cervico-thoracic spine in addition to being unable to see in front. Some mild and linear contractures of the neck can be managed by Z-plasty. Most cases are diffuse and need release (with or without excision) and skin cover. These patients pose the problem of endotracheal intubation to the anesthesiologist. In earlier times, blind nasal intubation was tried under ether and the success depended on the competence of the anesthetist and the luck of the patient. If it failed, the surgeon used to help his colleague by releasing the contracture under local anesthetic infiltration. The release was also sometimes attempted under ketamine especially in children and intubation attempted. Once the intubation was done, complete release was done and skin cover provided. With the availability of laryngeal mask airway (LMA) and fiberoptic intubating bronchoscopes and the technical know-how, anesthetizing a severe post burn contracture has got simplified and safe. The patient is positioned by placing a large positioner under the shoulder and a stack of 2-3 positioner rings under the head. The neck contracture is released at the point of maximum tension and the extra rings gradually removed from under the head so that finally the patient's neck becomes hyperextended at the time of skin cover. The severe mentosternal contractures may need release from one mid-lateral line to other. The strap muscles may also need division to gain better release and contours. Any unsightly scars or infected skin pockets are excised and complete hemostasis achieved. The severest mentosternal contractures may open up an extensive area.

For the last several decades, a controversy repeatedly comes up with the publications in literature and presentations in burn conferences as to the superiority of skin flaps and split skin grafts over one another for post burn neck contracture management. As has been said above, there is no absolute indication for skin flap application as the bed is excellently graftable. The reasons given for use of flap cover are: (1) It does not require postoperative splintage as it does not contract, (2) it is one stage, (3) it has better color/texture and hence better esthetic result, (4) long term follow up is not required.

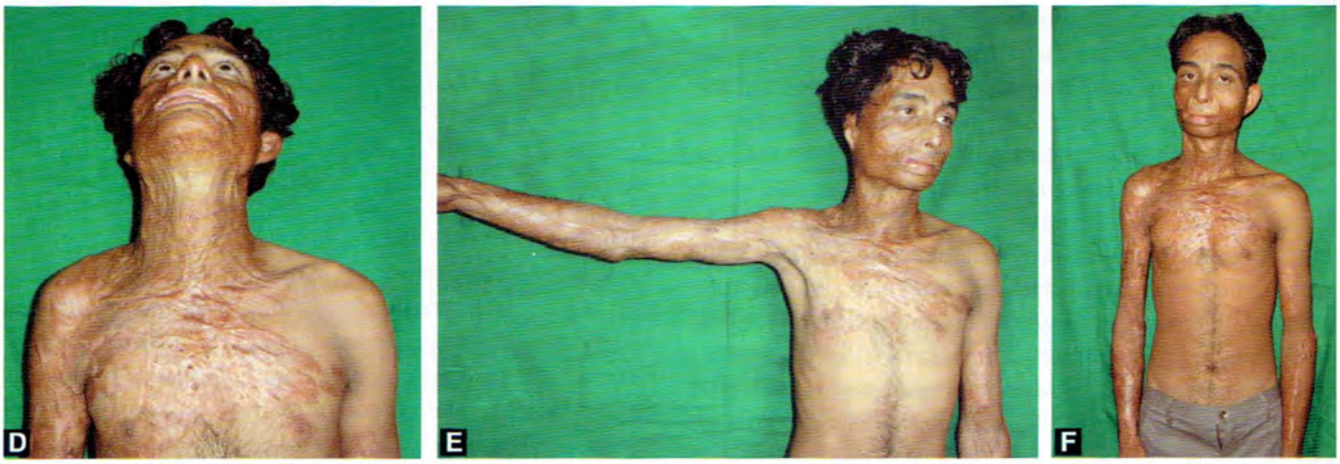
After going through the published literature and the photographs therein (without doubt the best results of the author(s) of those articles), the following facts become obvious to the present author (The author has intentionally omitted the references). (1) The main aim of the operation was excision of the post burn scar in the neck (which had a mild element of contracture) rather than a severe mentosternal contracture; (2) Even when the contracture was very severe, no importance was given to the scars on the surrounding areas of lower face, chin, shoulders and upper chest; (3) The healthy normal skin of the flap stood out conspicuously from the surrounding scarred skin like an island in the ocean. This appearance of the patient can be compared to that of a poor beggar wearing a highly expensive neck-tie! Consequently, the patient as a whole does not have a pleasing appearance; (4) The donor site morbidity of the flap itself was never taken into consideration; (5) Any flap, even a relatively thin one, tends to conceal the details of the neck contours and gives the neck a 'milk can' appearance. When free flaps are used, the circular suture line contracts and gives the flap a 'biscuit' or 'cookie' appearance obscuring the underlying contours further. When local pedicle flaps are used, they may become very thick even after many years when the patient gains weight spoiling the initial good result; (6) The flaps may be hairy depending on the donor site as well as the 'hairiness' of the individual patient. While presence of hair is obviously undesirable in females, it is no good either even in males because of totally different appearance from the normal male in shaven as well as non-shaven state; (7) All flap operations, including free microvascular flaps, were only theoretically single-staged. Actually they required 2-4 thinning procedures in addition, to get the desired contours, over a period of time requiring frequent patient visits. Some flaps were raised after tissue expansion. (8) Most of the articles are case reports of a few patients; (9) Secondary graft contraction in split skin grafts and the need for splintage, pressure garments, massage etc. have been presented as something highly unmanageable. This is not true; (10) In the unfortunate event of a pedicled flap failing partially, use of a split skin graft to close the resultant wound leads to a very poor esthetic result. Failure of a free flap will necessitate the use of large sheets of skin grafts which may not be now available.

The present author has exclusively and extensively used skin grafts for management of post burn mentosternal contractures over the last 23 years in a center with a great workload of post-burn sequelae and special interest in treating them and has achieved excellent results. He has the following reasons for using split thickness skin grafts for diffuse neck contractures (Figs 34.14A to D and 34.18A to 34.21D). (1) As a rule, it is a single-staged procedure, rarely requiring a second stage to improve the result; (2) The graft fits better into the surrounding post burn scars and grafted raw areas without any sudden changes in color and texture etc. (3) There is minimal donor site morbidity; (4) Far less operating time is required; (5) Excellent contours with finer details can be achieved; (6) It is suitable for mild as well as the most severe cases (7) Microsurgical skills and operating microscope are not required; (8) In the unfortunate event of a partial graft loss, minor graft losses heal by conservative management whereas bigger areas, even if grafted again, do not spoil the esthetics of the final result; (9) Most patients have multiple contractures, scars, losses and other reconstructive needs out of which neck is the first major one to be addressed (next only to upper eyelid). Improvement after neck release tremendously improves the appearance of the patient and his/her confidence in the treating surgeon. Hence, there is no question of poor compliance or lengthy follow up; (10) The contractures from as a result of full thickness burns which were not treated by split skin grafting in acute initial stage. After release of contracture neck, a split skin graft only is required; (11) Last but not the least, a split skin graft only can take care of the more than one billion populations. Thus, while more and more articles with newly discovered/modified old flaps will continue to appear in journals, the massive load of burn victims will continue to be benefitted only by the simple skin graft. It must be made amply clear to the reader that the author is not "anti-flap" and has no objection to treat the patient shown in Figures 34.22A and B using an appropriate flap as the scar-contracture was limited, surrounding region normal and overall other reconstructive needs nil.

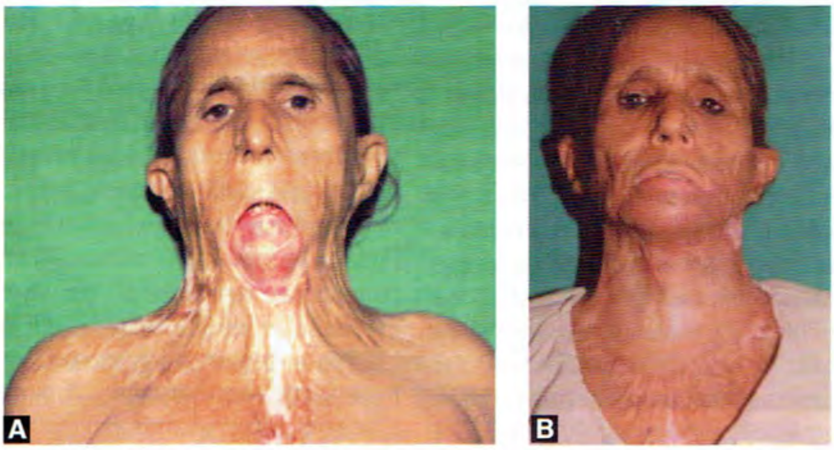
The sheets of skin grafts are applied with their junction lines horizontally placed and immobilized using tie-over sutures. Postoperatively, after the graft is taken up, the patient



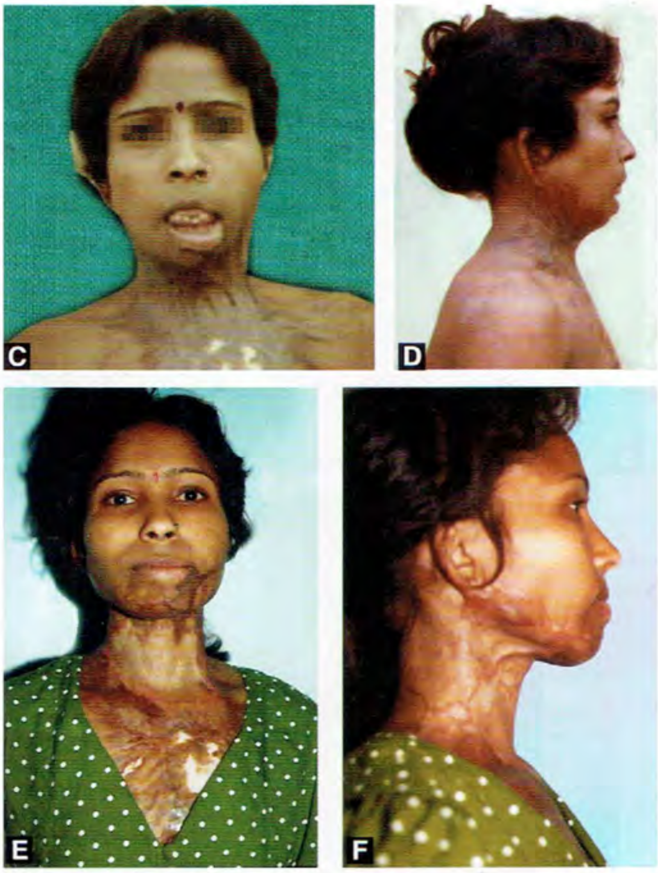
Figs 34.18A to C: (A and B) A 7-year-old boy with severe contracture of neck, right axilla and both elbows along with ectropion of lower lip with microstomia and extrinsic ectropion of lower eyelids. (C) A timely sequential release of all contractures



Figs 34.18D to F: Follow up at 18 years of age



Figs 34.19A and B: 50-year-old female with severe mentosternal contracture with completely everted lower lip. (B) Result after 1 year with adequate lip seal (Pre and Postoperative view)

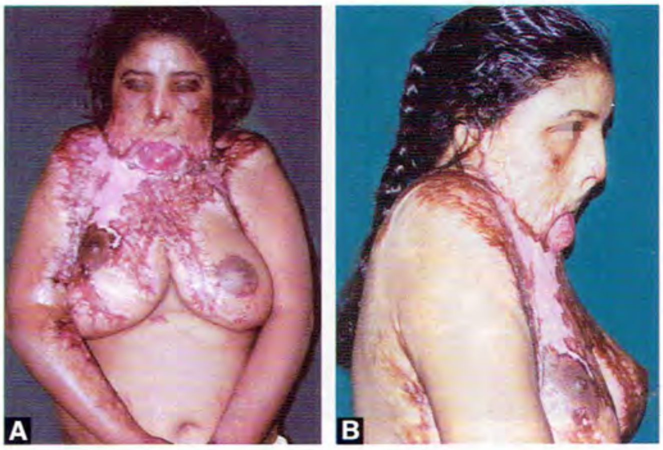


Figs 34.19C to F: Front and lateral view of post burn contracture neck in a 23-year-old female. Note the deformed lower teeth. (E and F) Postoperative result with complete restoration of cervicomental angle and lip seal after 1 year

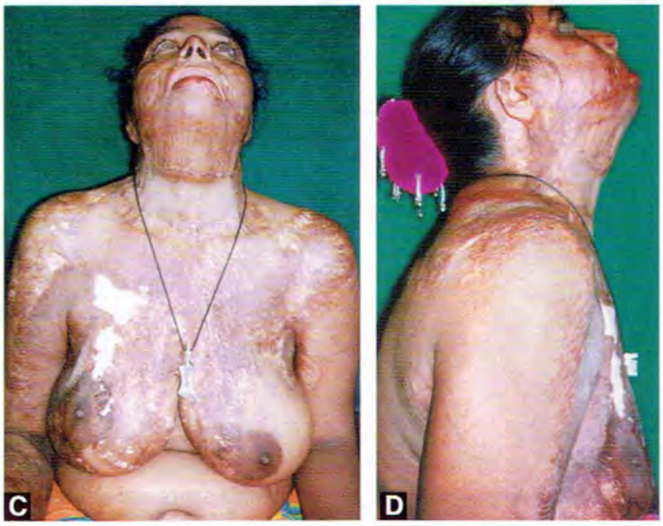
is given simple neck collars and chin-neck pressure garments and other care as detailed above for grafts. Patient's compliance in postoperative care of the graft is surgeon dependent. Better the surgeon in his doctor-patient relationship, better is the result!

Contractures of the Axilla

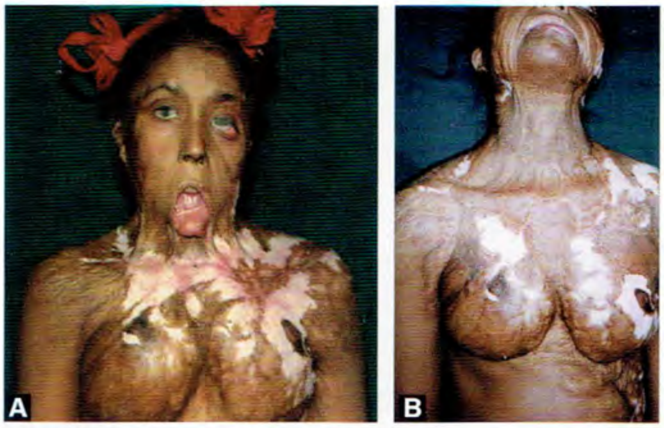
Contractures of the axilla are also very common. Similar to neck, the skin is thin and loose and the patient tends to keep the arm adducted. A contracture of the axilla may involve only the anterior or posterior axillary folds or there may be a diffuse contracture involving the whole of axilla (Figs 34.17A to 34.18F and 34.20A to D). Sometimes, the anterior and posterior folds are extensively involved but the normal hairy skin area in the apex of the axilla is spared. In the latter case, while releasing the contracture, the incision has to be made in such a way that neither the normal skin of the apex of the axilla is divided nor its anatomical position altered. Once the contracture is released, thick split skin graft is applied with tie-over sutures and the arm splinted with an aeroplane splint with arm abducted at an angle of 110 degrees. This splint is continued till the graft is mature and there is no risk of graft contraction. In selected cases, scapular or para scapular flap, if available and sufficient, can be planned preoperatively to provide cover and may obviate the need of postoperative splints which are cumbersome. Sometimes, patients present with axillary contractures which are linear webs and a single or multiple Z-plasty or other multiple inter-digitating flaps-plasty (e.g. V-M plasty) may be designed and contracture treated.



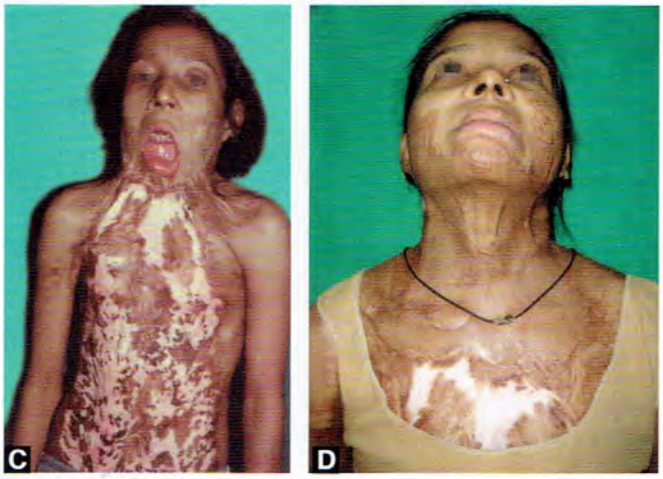
Figs 34.20A and B: 25-year-old female with severe contracture of neck, right axilla, right elbow and breasts



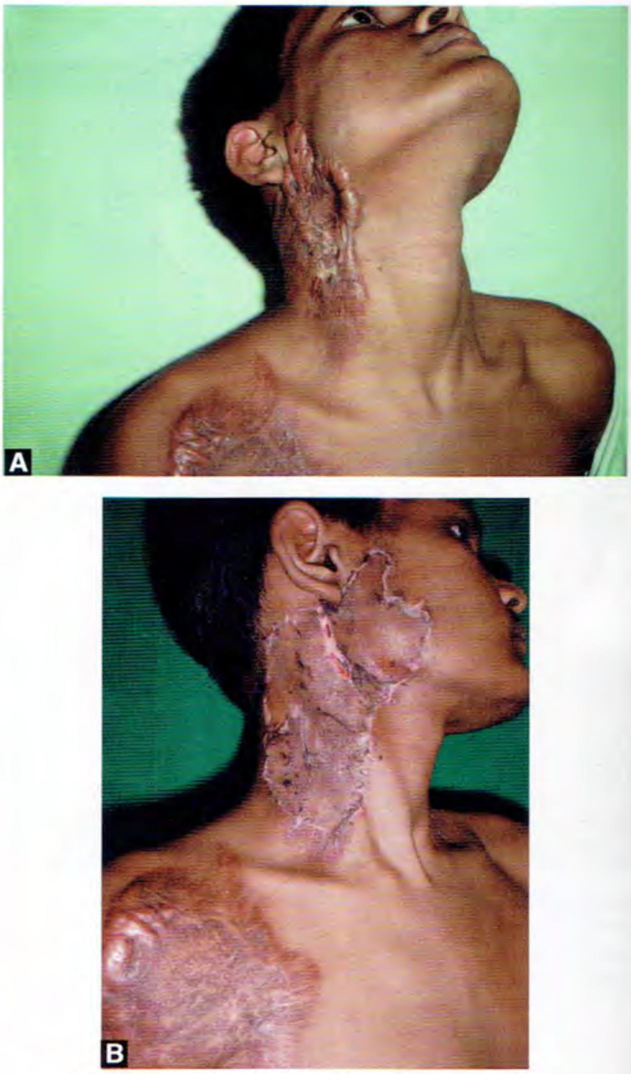
Figs 34.20C and D: 2 years after release and split skin grafting. Note the improvement in breast contours without any surgical intervention on breast



Figs 34.21A and B: Severe mentosternal contracture with ectropion lower lip and pulled up breasts. After release and split skin grafting, full range of movement achieved. Good contours of the neck



Figs 34.21C and D: 9-year-old girl with severe cervicomental contracture and depigmented scars on anterior trunk. Postoperative result after 8 years. Release of contracture and split skin grafting done

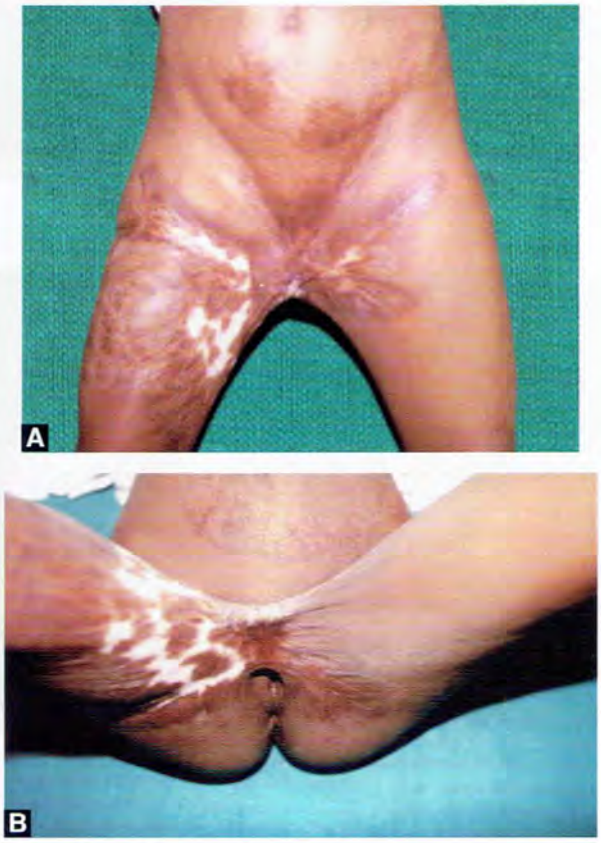


Figs 34.22A and B: Hypertrophic scar lateral neck with mild contracture. Pre and early postoperative result (See text)

Flexion Contractures of the Elbow (Cubital Fossa), Wrist and Knee (Popliteal Fossa)

Flexion contractures of the elbow (cubital fossa), wrist and knee (popliteal fossa) are similar in many respects. A severe contracture developed in any of these sites in early childhood and coming for corrective surgery after many years in adulthood is very difficult to correct completely because the shortened musculo-tendinous units and neurovascular structures stand out as bow-strings and any further attempt to straighten them forcefully may injure and damage them. Moreover, the joint may be subluxated or deformed so as not to allow the limb to straighten despite complete release (Figs 34.8A to D and 34.12A to E). In these cases, after maximal possible release, the raw area is splinted with a Crammer wire splint which is gradually straightened over a period of 10-14 days and then split skin grafted. Here again, the release incision is given opposite the point of maximum tension, usually opposite the joint line. Since rigid post operative splintage (to counter secondary graft contraction) is easier in a straight limb, an intermediate thickness skin graft can suffice for excellent results (Figs 34.18A to F). In bilateral elbow contractures, both sides should not be operated together to allow the patient some self reliance. However, bilateral knee contractures should be operated simultaneously to make the patient ambulatory as soon as possible.

Contractures of groin, when unilateral, may cause difficulty in normal standing/walking. When they are bilateral, the contracting band usually goes from one thigh across groin and pubis to the other groin and thigh leading to difficulty in abduction of thighs **(perineal contractures)** (Figs 34.23A to C).



Figs 34.23A and B: Bilateral groin contracture

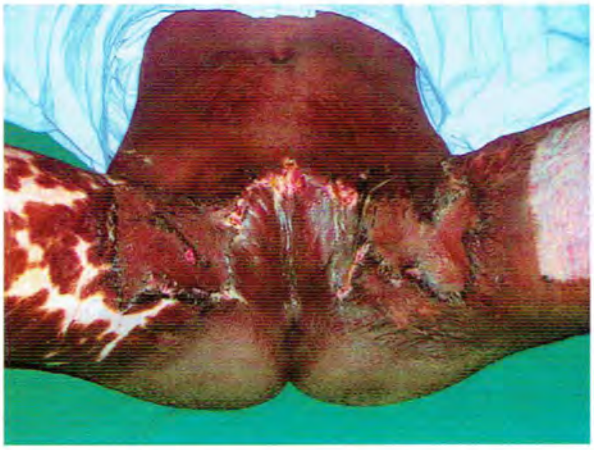


Fig. 34.23C: After release and split skin grafting

The **breast** is discussed in detail later in this chapter while contractures and deformities of hand are devoted an entire chapter elsewhere in this book.

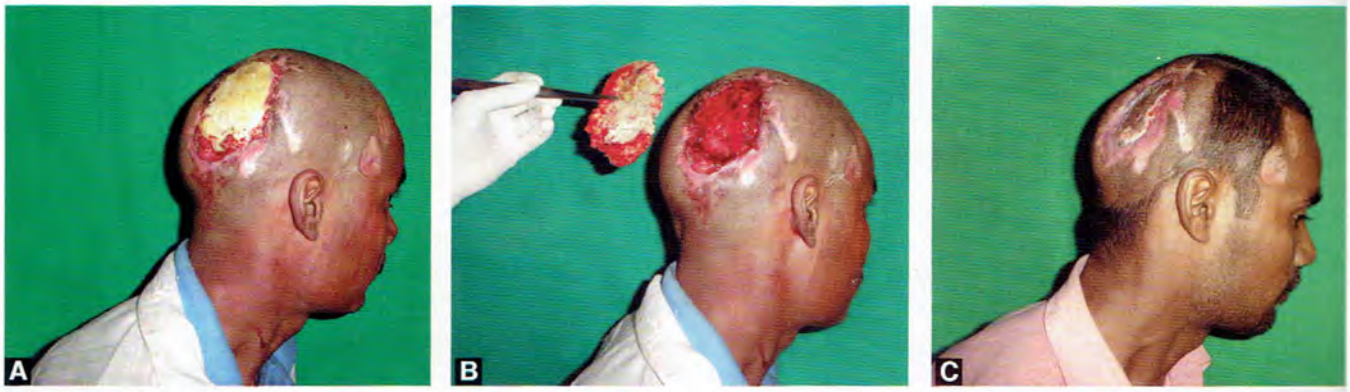
POST BURN LOSSES

Burn victims may be left with losses of one or more specialized structures or body parts which may be either extremely difficult to reconstruct or else may not be possible to restore. Thus, there may be loss of scalp, pinna, eyebrows, nose, moustaches, nipple-areola complex, penis and testis etc. Amputations of digits or limbs at various levels, especially after electrical burns and loss of testis, are almost beyond the realm of reconstruction. They need prosthetic restoration. Loss of thumb may be treated by reconstruction with one of the many available techniques. Loss of vision is very common after chemical burn injuries to eye.

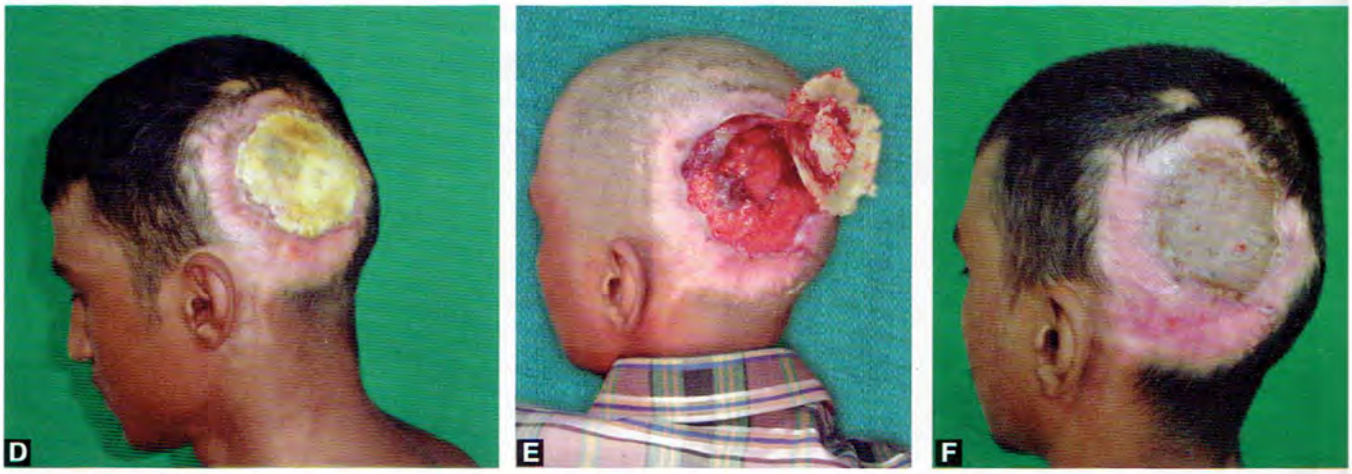
These losses are also dependent on the depth of burn. However, they may sometimes be prevented, e.g. by timely escharotomy in full thickness circumferential burns of limbs or digits or by prevention of chondritis (with eventual loss of pinna) by appropriate management of burns of pinna in the acute stage.

Scalp

Deep dermal thermal burns of the scalp often heal with alopecia patches due to damage to hair follicles and scarring. On the other hand, electrical burns of the scalp often involve full thickness of the scalp and even the calvarium. These wounds do not heal without appropriate surgical intervention. The initial intervention may leave the patient with patch of split skin graft over the calvarium. These patients later present for restoration of hair bearing scalp. A number of patients reach a plastic surgeon after long periods of 6 months to 2 years or more following electrical burns of scalp. They have a chronically exposed calvarium.[33] Typically, these patients had no access to specialized treatment of burns and were treated by dressings alone. Their wounds heal by formation of multiple contractures. However, since contraction is limited on rigid skull, they are ultimately left with an area of exposed calvarium with dried up outer table of skull or sequestrum formation (Figs 34.24A to F). The patients with chronically exposed calvarium should be treated by removing (chiseling out) the sequestrum of the outer table of skull and wound closure by application of a split skin graft over the diploe. Only very small wounds can be closed primarily using local rotation flaps in the same stage.



Figs 34.24A to C: (A) Chronically exposed calvarium (15 months post burn). (B) Sequestrum removed exposing underlying granulation tissue. (C) 4-weeks later, healed with split skin graft

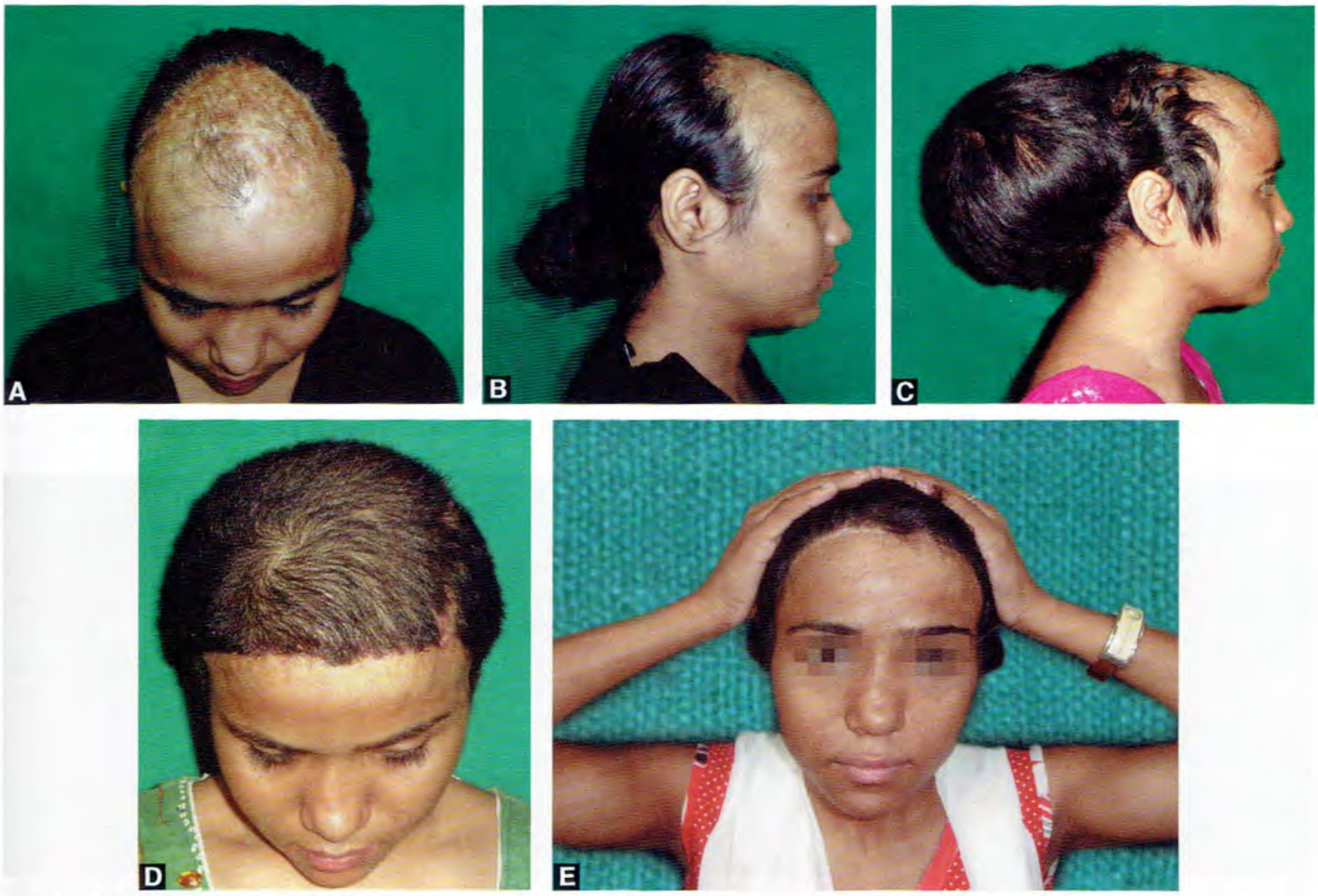


Figs 34.24D to F: (D) Chronically exposed calvarium (12 months post burn). (E) Sequestrum removed exposing underlying granulation tissue. (F) 3-weeks later, healed with split skin graft

Transposition flaps are absolutely contraindicated in these situations as they leave the alopecic areas in the form of split skin graft over donor site and greatly jeopardize the adjacent areas which may be needed later for tissue expansion for ideal scalp restoration.

The restoration of hair bearing scalp has been revolutionized over the last 25 years with the introduction and availability of tissue expansion and expanders respectively (Figs 34.25A to 34.26E). In earlier times, the aim was only to restore the anterior hair line using one of repeatedly delayed strips of available hairy scalp. Presently, the surgeon tries to excise all of non-hair bearing areas. Except for very small alopecic areas which can be excised and primarily closed either by simple undermining and advancement or by local rotation flaps after triangulating the defect, all other larger areas are treated by tissue expansion.

It is the author's practice to get scalp completely shaved at the time of insertion as well as at the removal of expander. The former has shown many unexpected scars in the scalp which have changed the plan of tissue expander placement. This also allows proper part preparation which is so very essential when one is inserting a foreign body. It is also possible that the ideal size and shape of the expander is not available (very common in a developing country). In such circumstances, use of a less than ideal expander may give excellent results if the entire scalp is shaved and the expander has been inflated a little more than the calculated amount. At the time of removal one may have to change the plan of movement of the expanded tissue because the expansion did not proceed in the desired direction. It proves handy if the whole scalp is shaved and prepared. The tissue expander is inserted in subgaleal plane. The incision should be in the healthy skin, 1.5-2 cm away from the scar/graft with



Figs 34. 25A to E: (A and B) Front and lateral view of post burn cicatricial alopecia in 20-year old girl with severely recessed anterior hairline. (C) Tissue expansion in progress (over 15 weeks). (D) Complete coverage of scalp with expanded tissues. (E) Patient trying to show complete restoration of normal anterior hairline

healthy skin junction, to avoid breaking down of the incision at the time of expansion. The port should be as far away as possible to avoid overlapping with the expanding implant and getting punctured unintentionally.

The incision is closed in two layers with deeper layer of polyglactin910 and skin with prolene. A small amount of sterile normal saline is injected at the time of insertion to flatten out the folds in the expander.

After a period of two weeks, once the wound has healed, gradual inflations are started. The inflations are done biweekly till the required scalp has been generated, usually a period of 3 months. The amount injected in any one sitting should neither blanch the overlying skin nor should cause undue discomfort to the patient. A record must be made of the dates and the amount injected. One must also observe for any thinning of the suture line or the skin being expanded. The expansion may be stopped for a while till recovery occurs and then, proceed slowly and cautiously. Strict asepsis and anti-septic precautions must be taken at all times to avoid the complication. After complete expansion has been achieved, the expander is removed through the old incision and expanded skin moved over the alopecic area.

Eyebrows[34,35]

Facial burns are very common and so are the burns of eyebrows leading to loss of hair bearing supraorbital region. The loss of eyebrows may be unilateral or bilateral. Also it may be complete, i.e. no hair is left or it may be incomplete, i.e. a significant number of hairs may still remain. Without eyebrows the face looks bland. However, the eyebrow reconstruction is usually the last operative intervention on the patient. This is because their restoration is purely aesthetic in nature and they are to be restored only after the scars and contractures of the surrounding facial region have

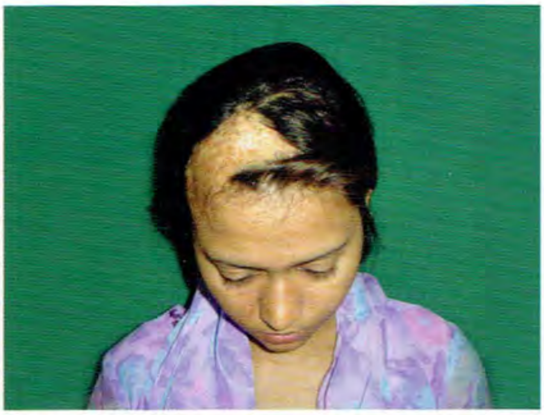


Fig. 34.26A: Post burn alopecia in 23-year-old girl

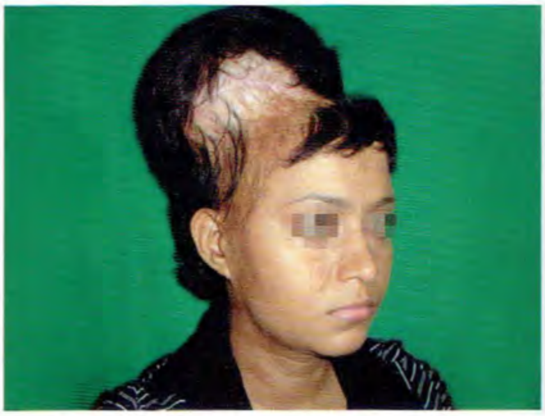
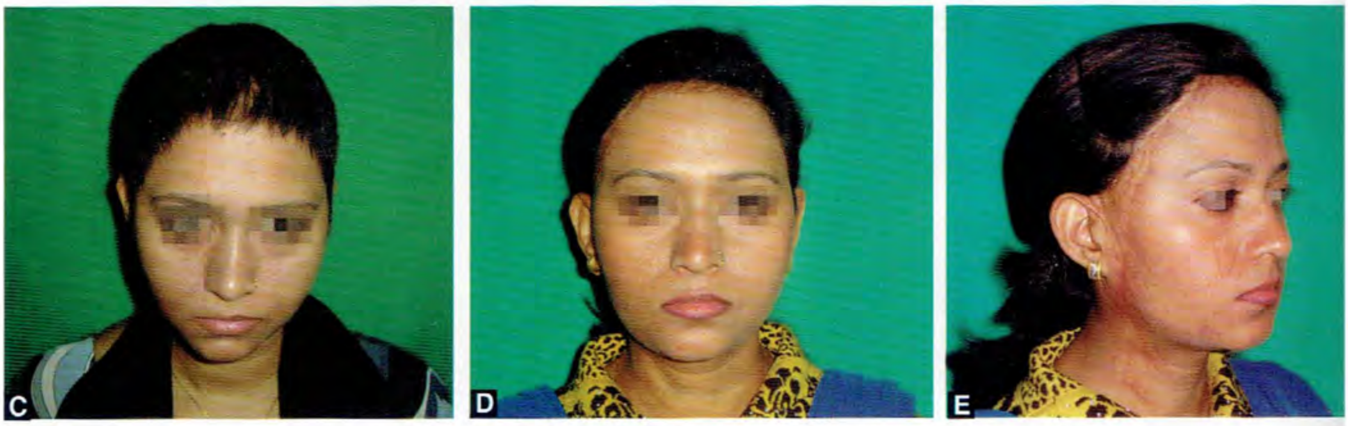


Fig. 34.26B: Tissue expansion in progress



Figs 34.26C to E: Final result with complete restoration of scalp hair

settled well so that the reconstructed eyebrows do not change their position. Thus, one can rightly term the eyebrows restoration as providing a 'finishing touch' to face. In the intervening period, the patient is advised to use an eyebrow pencil. Many patients, especially females, may opt for continued use of pencil rather than go ahead with reconstructive surgery. In this non-operative option the patient has to take the trouble of reconstructing the eyebrow daily. In the surgically reconstructed eyebrow as well, the patient has to take the trouble of regularly trimming the hair as they grow. This gives a waxing and waning of the eyebrow density and bushiness as compared to the uniformity of eyebrow pencil use. The male patients, however, should always be offered surgical reconstruction.

A number of operative techniques are available to restore the eyebrow when a normal healthy skin is available in the region. In post burn cases, however, the brow region is generally scarred or split skin grafted and a pedicled flap from the scalp is ideal. It must be added here that excisional surgery is absolutely forbidden in this region in acute stage, even if the burn appears full thickness as the eyebrow hairs have very deep seated roots and may survive the injury giving reasonable result.

An island pedicle flap from the scalp based on the ipsilateral superficial temporal artery (posterior branch) is marked. The position of artery is found by light palpation and confirmed by Doppler, if available. Planning in reverse is done to mark the skin island on the scalp. The flap can be raised in two ways. The skin island is raised on an almost skeletonised superficial temporal artery and vein, and then transferred to the supraorbital region with the pedicle being tunneled through the intervening skin from the pre-auricular region to the recipient site. This method always has a danger of partial or complete loss due to island being not supplied or drained adequately or compression of the pedicle in the tunnel. A safer, although two-staged, technique followed by the author is to take a broad pedicle artery and vein and intervening fascia with the skin island being 1.5 to 2 times the width of desired eyebrow and transferred over intact skin with interpolation. This is an excellent and safe technique for the novice or the surgeon who performs this operation only occasionally. Since the initial inset is hundred percent, the pedicle is divided without any delay at the end of 3 weeks. Once the tissues have settled completely after a period of several months, the desired eyebrow width and shape are marked at appropriate place on the hairy island and excess excised. The newly created eyebrows require frequent trimming for esthetic appearance (Figs 34.27A to 34.28B).

In patients where the eyebrow loss is partial or else the supraorbital skin is reasonably healthy and the surgeon has a feeling that hair grafting may be successful, it may be attempted. One may do the older techniques of strip grafting (not preferred due to high failure) or the newer techniques of micro-follicular or single hair grafting as one does for male pattern baldness of the scalp.

Nose

The nose occupies the most prominent position on the face making it vulnerable to almost every burn injury involving the upper part of the body and there may be associated inhalational injury as well. A partial thickness burn injury can lead to scarring of the skin of the nose with essentially preserved shape. It may lead to narrowing or eversion of

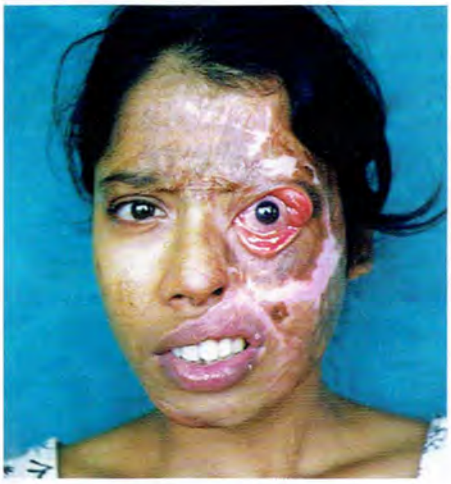


Fig. 34.27A: Patient with hypopigmented scars on left face , ectropion of left upper and lower eyelids and left half of upper lip with loss of ipsilateral eyebrow

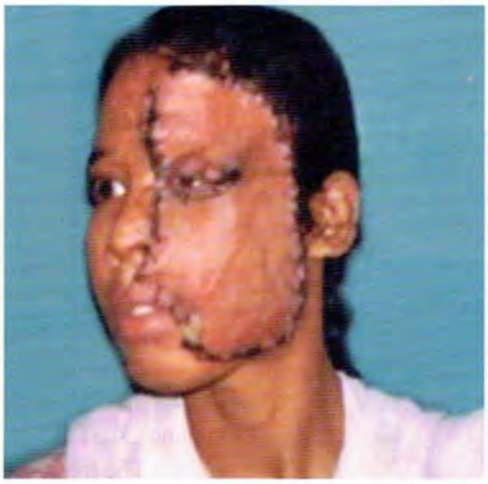


Fig. 34.27B: Appearance after release of ectropion of both lids and lip and excision of scar and cover with a single sheet of graft from thigh in one stage. At 8 days

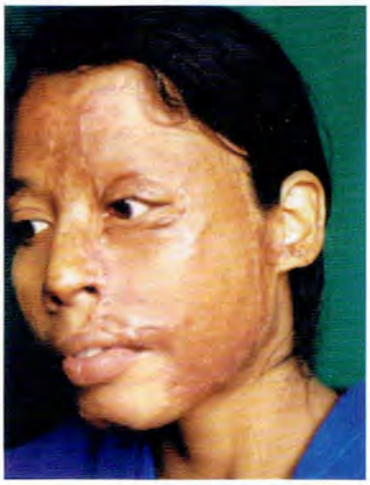


Fig. 34.27C: At 6 months

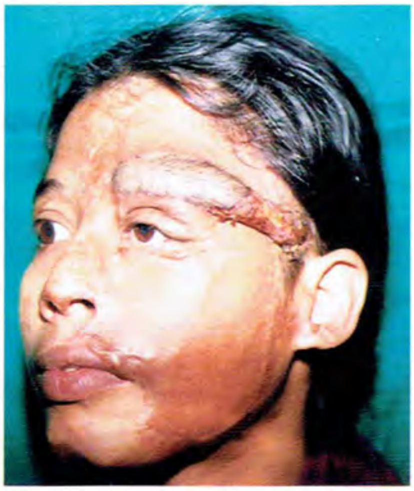


Fig. 34.27D: Eyebrow reconstruction being done by pedicled superficial temporal artery flap

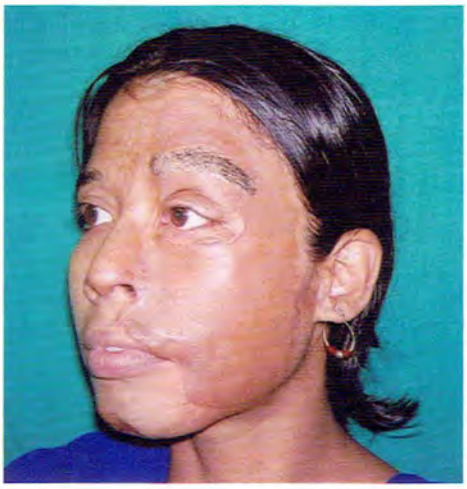


Fig. 34.27E: Final result—2 years follow up. Well settled graft and symmetrical eyebrows

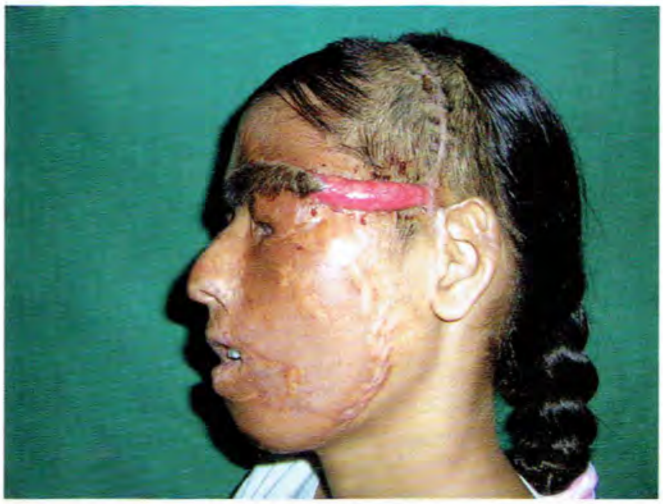


Fig. 34.28A: Eyebrow being reconstructed by pedicled flap from scalp. Note the width of flap

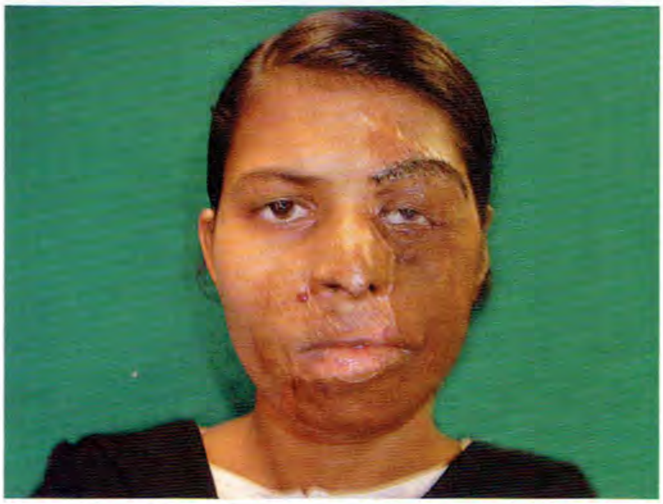


Fig. 34.28B: Result after trimming of flap and hair

external naris (nasal stenosis or ectropion respectively). A full thickness burn of skin or a still deeper injury may lead to a subtotal loss of nose, requiring lining, support and cover for its reconstruction. Total loss of nose, i.e. loss of bony framework is extremely uncommon. The restoration of nasal structure, function and cosmesis is vital to restore the self esteem of the burn victim. Here again, whole of the plastic surgeon's armamentarium for reconstructive rhinoplasty in various permutations and combinations along with his ingenuity is needed to achieve the goal of 'return to society'. In most of the patients who come for reconstructive rhinoplasty to a plastic surgeon, the cause is either a human bite or other trauma or else they have a post-excisional (malignant tumors) defect. The reconstructive options of using forehead flap or nasolabial flaps which give excellent results in these patients are generally not available in burn patients. Commonly, the less than ideal options of restoring the nose from medial arm or other distant regions have to be chosen.

In patients with scarred nasal skin cover only, the latter can be replaced as a whole with a sheet graft or expanded thin skin from the forehead, if available. The aim should be a result that mixes well with the surrounding face which has healed spontaneously or after application of skin grafts or flaps, etc. Before this excision of scar is done it is preferable that the surrounding scars/grafts/flaps have well settled and will not cause any extrinsic pull on the nose. Thus, ectropion of the eyelids and upper lip, microstomia, tight skin of the cheeks, bands passing from nose to cheeks, etc. should have been corrected. The scar should be excised thinly preserving whatever subcutaneous tissues to prevent a 'skeletonised' look to the nose. When cover is being provided with a graft, the incision at nasofacial junction must be slightly on to cheek as well as with a slight bulge towards the face to take care of secondary graft contraction. One must try to feed excess of graft as well. These measures prevent the junction line between nose and face to come over the nose and spoil the outcome. The graft can be harvested from the medial arm or groin (Figs 34.4A to D and 34.31A to C).

The management of nostril stenosis is difficult and every effort should be made in the acute stage for its prevention or minimization by using moulded splints till scar maturation. Once the scars have matured, the stenosis should be corrected by excision-release of scar and use of inter-digitating or local flaps and thick skin grafts. The ectropion of the external naris, again, needs release and cover either with thick skin graft or local flaps, may be from the nasolabial region, if available. Whenever grafts are used at free margin of the nose, postoperative moulds must be worn by the patient till graft maturation.

The patients with subtotal loss of nose require recreation of the defect and assessment of the available donor site(s) for provision of lining, cover and support. For smaller defects, local flaps from nose or nasolabial region, if available, can be used. For larger defects forehead skin, if healthy and sufficient, can be used for cover with lining being provided by turn down flaps. They do not need a support as the forehead tissues are sufficiently tough to maintain their shape. Forehead flap rhinoplasty using one of its various modifications like midline or oblique forehead flap or after tissue expansion is commonly done procedure performed by every plastic surgeon for non burn indications. Detailed techniques are available in all modern plastic surgical books.[36]

Very commonly, however, a patient with post burn loss of nose does not have the forehead tissues fit for use and it is in these patients that the medial arm tube has to be constructed and transferred in stages for reconstructive rhinoplasty. In cases where this is also not possible, other donor sites like groin or abdomen have to be considered for constructing the tube. To avoid multi-staged operations and their attendant morbidity, microvascular free tissue transfer has been done by various workers using flaps based on radial or dorsalis pedis artery, etc.

The detailed technique of arm tube rhinoplasty is not covered in modern plastic surgical books as it is considered old-fashioned and outdated (Figs 34.29A to 34.30D). Also it does not give as good results as forehead flap. The author has, however, found this donor site to be an excellent source in those burnt patients where forehead is scarred or grafted. Although multi-staged in itself, one must remember that construction of tube may not add any stage as it is usually combined along with some other reconstructive need of the burnt patient. The only real morbidity is for a period of three weeks when the first inset is done on the nose and the arm has to be fixed to head. The delay and division of the tube flap from the arm, inset on the nose and return of pedicle to arm can all be done under local anesthesia. The skin of the medial side of the arm is chosen as it is fair-colored, non-hairy, easily covered by elbow level sleeves and is mostly in contact with chest wall and hence not visible.

Two parallel incisions, about 8 cm. apart and 16 cm long, are made in the long axis of the arm and deepened till

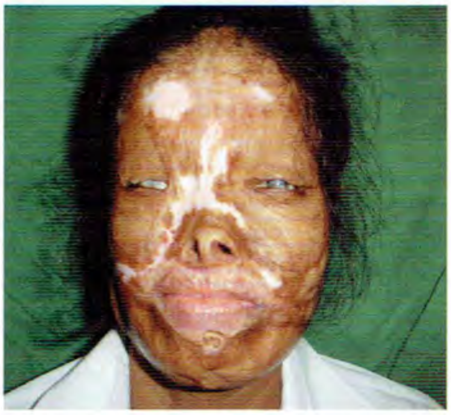


Fig. 34.29A: Young adult female with post burn extensive facial scarring, subtotal loss of nose and complete loss of vision both eyes due to homicidal acid burns

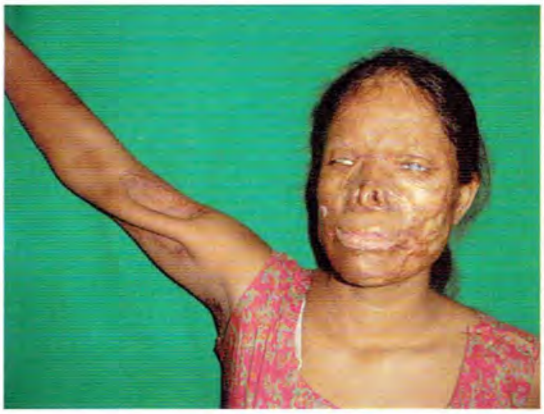


Fig. 34.29B: Result after resurfacing of forehead, release of upper lip ectropion and arm tube construction

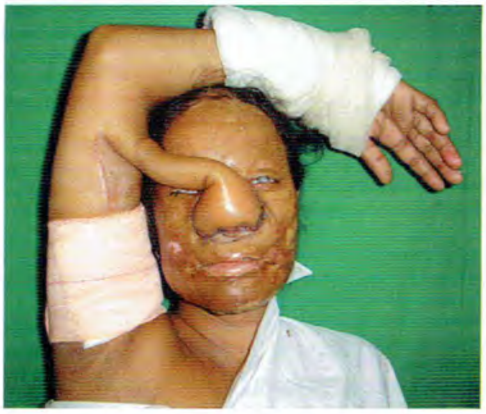


Fig. 34.29C: Arm tube inset on to nose

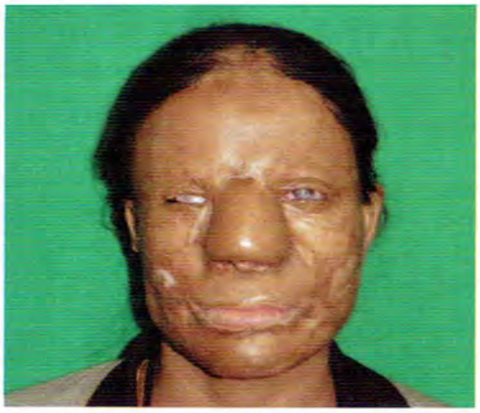


Fig. 34.29D: Final result after nasal reconstruction

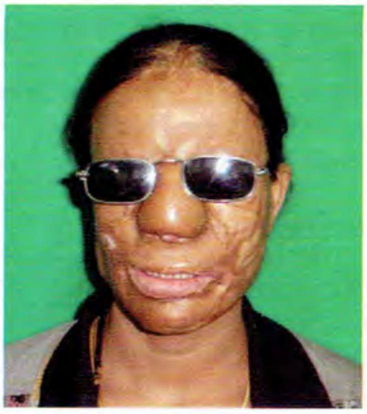
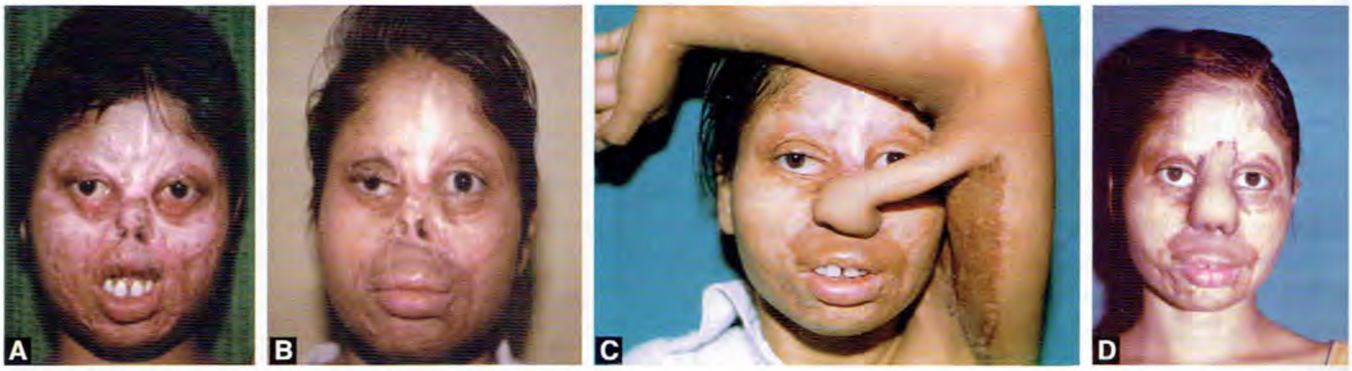


Fig. 34.29E: Successful return to society



Figs 34.30A to D: (A) 20-year-old female with bilateral ectropion of eyelids and upper lip, subtotal loss of nose and complete loss of both eyebrows. (B) After release of upper lip and eyelids. (C) Arm tube inset on nose. Note the small and scarred forehead. (D) Nasal reconstruction completed

the whole of subcutaneous tissues. Undermining is done in this plane and a bipedicled, random pattern skin flap (2:1 ratio) is raised. The longitudinal cut margins of the flap are stitched to each other forming a sort of suit-case handle. It is not possible to stitch the ends of the tube and triangular raw areas on the under surface of the ends of skin tube and the donor bed are split skin grafted with a single sheet graft using tie-over sutures. Once the wounds have healed and the graft taken up, the tube is allowed to 'mature' and become soft and supple over a period of 10-12 weeks. The proximal end of this tube is, then, delayed by a transverse incision and the first inset on to nose is done in 7-10 days. The lining is provided by turn over flaps from the defect margin and/or from the nasolabial flaps. The tube is opened at the seam for the required length and the first inset is done to form the ala, the columella and the lower part of the nose (as much as possible) as described by Sawhney (1981).[37] The width of the adult male nose is around 7.5 cm at the broadest part; hence the width of tube was kept at 8 cm to allow for tissue loss in scarred seam. At the end of 3 weeks, the tube is divided at appropriate level keeping the tissues required on the nose. Final inset is done on the nose and pedicle is returned back after excising the skin graft on the donor site equal to the length of pedicle available for return.

Moustaches[38-42]

When a patient presents with a severe ectropion of the upper lip, a release and thick split thickness skin graft application may restore the normal appearance in a child or an adult female. However, in adult males, (Figs 34.31A to C) absence of the moustaches may be resented and one may request for its restoration to have a more natural and masculine appearance.

Hair-bearing skin to upper lip can be transferred from the scalp or from the neck. Very few papers with case reports only have been published in literature about moustache restoration in male burn patients. A bi-pedicled flap from either of these sites can be transferred as a 'visor' flap('bucket handle').The neck flap needs appropriate delays as they are not axial pattern and do not contain any named vessel in their pedicles. However, a scalp flap may be raised on both superficial temporal vascular pedicles and needs no delay. An L-shaped pedicled flap based on a single superficial temporal artery incorporating the parietal and occipital scalp with primary closure of the donor site has been described to restore whole of the moustache. A free flap based on one superficial temporal artery or an occipital artery has also been described to restore the moustache with anastomosis being done with facial vessels.

Pinna

Reconstruction of pinna or auricle in patients of microtia is a challenging task for a plastic surgeon with a select few doing the job and producing reasonably good results. One can imagine the challenge when healthy skin cover of microtia is replaced by scarred skin in a burn patient. The results are bound to be far from satisfying the patient and the surgeon alike. The pinna is burnt as a part of extensive burn injuries involving face and upper parts of the body. The skin of the pinna is very thin with almost no subcutaneous tissue especially on its anterolateral aspect. The underlying cartilage is directly dependent on overlying skin for its nutrition. The pinna may be lost directly due to a very deep burn injury or else following chondritis of auricular cartilage leading to its resorption and deformation of the three-dimensional architecture.

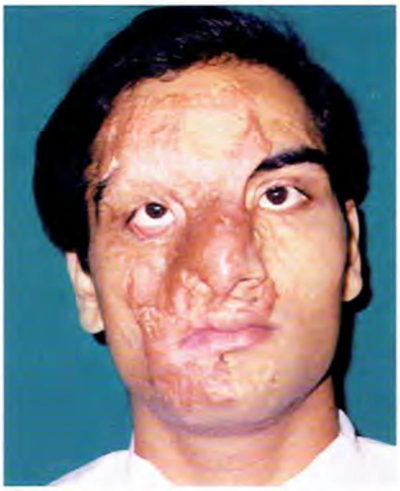
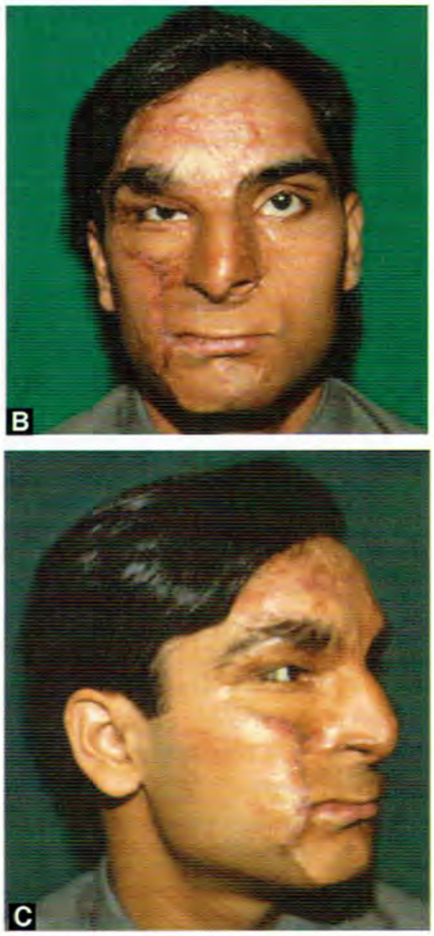


Fig. 34.31A: 23-year male with post acid burns hypertrophic scars on face and nose, ectropion of upper lip and loss of right eyebrow



Figs 34.31B and C: Release and split skin graft to cover the upper lip. Nose has been resurfaced with a skin graft as an esthetic unit. Eyebrow has been reconstructed from scalp flap (front and right lateral views)

The post burn deformities of the pinna may range from minor helical margin irregularities, loss of ear lobule, auriculocephalic adhesions to complete loss of pinna. In patients with loss of helical rim, skin tubes can be constructed in pre or post auricular region or even in the neck and transferred in stages. Loss of ear lobule can be dealt with one of the various techniques like Zenteno Alanis, etc. Adhesions between pinna and scalp need simple release and application of thick split skin graft.

The reconstruction of total loss of pinna[43,44] involves framework fabrication from autogenous costal cartilage or use of one of the various commercially available implants (Silastic, Medpore ® etc.) and provision of skin cover. It is the poor quality with respect to texture and elasticity and shortage of skin that makes the draping of costal cartilage framework or auricular implants extremely risky with ever present danger of their exposure and a catastrophe. Undermining of the scarred skin is dangerous in itself. With the availability of temporoparietal fascial flap, the problem of coverage has been solved to some extent. In case the skin is reasonably healthy and can withstand tissue expansion, the procedure can be done to regenerate sufficient skin in case of its shortage. Ear reconstruction is necessitated not only for esthetic reasons but also to support the spectacles. In India, wearing ornaments in the pinna is almost a rule in females of all age groups.

When pinna is reconstructed using an autogenous costal cartilage, the synchondrotic portion of the sixth, seventh and eighth ribs is harvested using extra-perichondrial dissection to obtain a complete graft. Cartilage framework fabrication is done with the help of a template and helical and conchal margins are augmented. If local cover is available, a thin pocket is created or else after an incision at the appropriate place, the framework or an implant is covered with temporoparietal fascial flap which, in turn, is split skin grafted.

Breast

Burns of the anterior chest region are very common in female children as well as adults. They are generally part of the extensive burns of the surrounding areas i.e., head and neck, abdomen, arms and axillae. In a prepubertal female child the mammary gland which is 4-8 mm. in size is located deep in the subcutaneous tissues. The bud is connected to the nipple by the mammary ducts which are lined by epithelium. Even in burns involving full thickness of skin, the breast bud gets spared and the proliferating epithelium of the milk ducts regenerates a nipple like structure over the developing mound. There may be lack of areola in such cases and the nipple may be hypoplastic and of the same color as of the surrounding skin. It is to be noted that complete lack of development of breast i.e., mound as well as nipple and areola complex (NAC) occurs only when the NAC region has been removed energetically but unfortunately during excisional surgery or else burnt deep up to the muscles in a prepubertal child as is possible in contact electrical burns (Fig. 34.32). Sloughing of NAC in a conservatively managed prepubertal female child does not mean that the mound will not develop at the time of puberty. Hence, any form of surgical excision or debridement is absolutely contraindicated in this region. These areas must be allowed to heal conservatively with repeated dressings and the raw areas should be split skin grafted after the granulation tissue forms over the region. After complete healing of the wounds, the grafted and spontaneously healed areas are taken care of as usual with massage, pressure garments etc. Surgical intervention of any sort is to be started only after the onset of puberty when the breast development begins.

Reconstruction of the Burned Breast[45-47]

Reconstruction of the burnt female breast is challenging because it affects the emotional well-being of a growing adolescent girl. Burns of the anterior chest wall in females can result in a variety of deformities which are dependent not only on the depth and extent of the burn but also on the age of the female victim at the time of burn. In patients who get burnt after the full development of the breasts, there can be deformed breast(s) with or without partial or complete loss of NAC. In prepubertal children, on the other hand, the problem may not even be identifiable fully and/or treatable till the child shows some signs of breast development at the time of menarche. In adult females, the presence of loose breast skin heals many raw areas with contraction without any need of skin grafting leading to "mastopexy" and sometimes better contours. This situation, if unilateral, may call for improving the unburnt side by appropriate operation to achieve symmetry of contours in consultation with the patient rather than "surgically deteriorating" the burnt side by releasing and inserting more graft. Also, in children, more stages are required as compared to adults due to continued growth of the breast mound resulting in graft falling short in amount again and again.

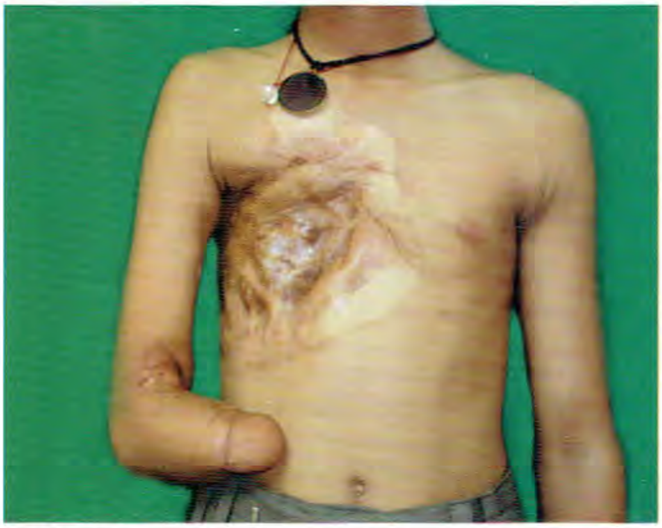


Fig. 34.32: Contact electrical burns of right anterior chest wall up to rib cage in a10-year-old girl with gangrene of right upper limb necessitating below elbow amputation. She needs breast reconstruction at appropriate time using myocutaneous flaps with silicone implant

Reconstruction of the Mound

The reconstruction of the breast mound in post burn cases differs greatly from that in post mastectomy cases. Whereas in the latter case, the breast has been totally removed, in post burn cases the apparent projection of the mound has been suppressed by the tight scar over the breast tissue. Thus, the bulk of breast mound is only apparently absent. It is actually present beneath the tight scar (Figs 34.33A to 34.36E). It is only in an extremely rare case that the breast was completely burnt right up to the costal muscles or when it was unfortunately excised completely by the surgeon during acute management that reconstruction of the mound is required. In these cases the techniques used for postmastectomy reconstruction like use of myocutaneous flaps, free or pedicled, tissue expanders and silicone breast implants etc. may be used. The author has no personal experience of this requirement in his 23 years of experience of working in the second largest burn center in the second most populous country.

In patients who have post burn contractures of the neck or axilla in addition, the breast reconstruction should begin only after the former are released and their grafts/flaps well settled. This is especially true in patients who have their contracting bands of breast, neck and axilla in continuity. The breast mound and its position improve considerably after the neck and axilla return to normal.

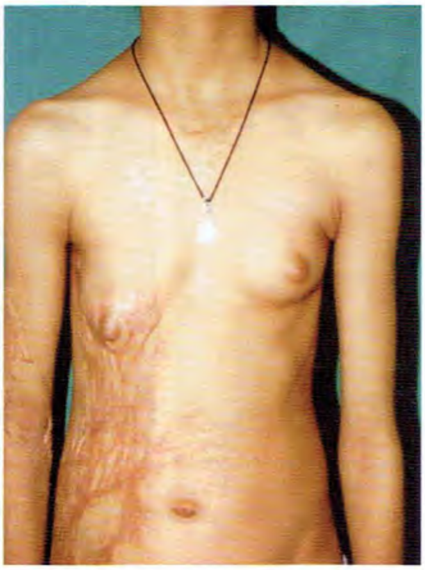


Fig. 34.33A: A 15-year-old girl with post burn contracture of right breast

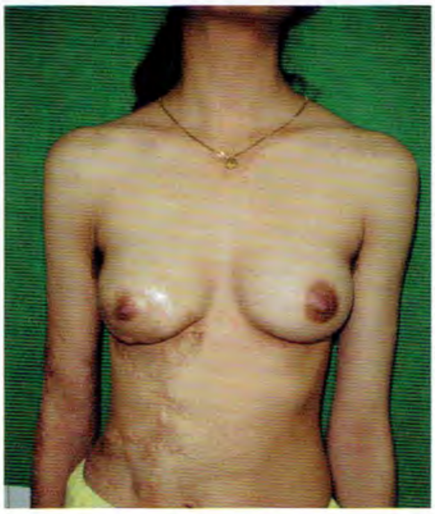


Fig. 34.33B: Release and split skin graft done achieving normal contours and size of breast (result after six years)

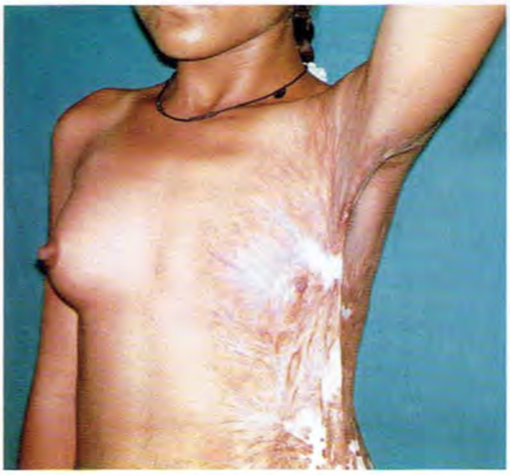


Fig. 34.34A: A 15-year-old girl with post burn contracture of left axilla with apparently absent/undeveloped breast mound

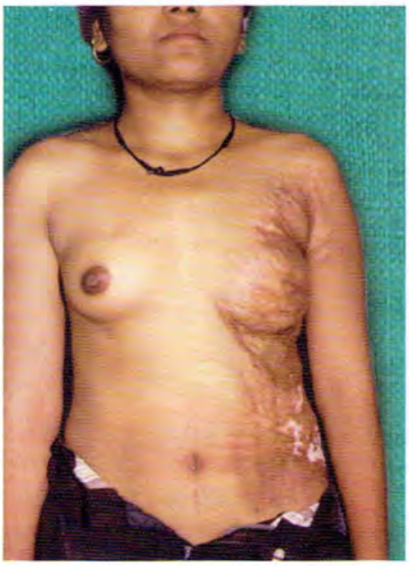
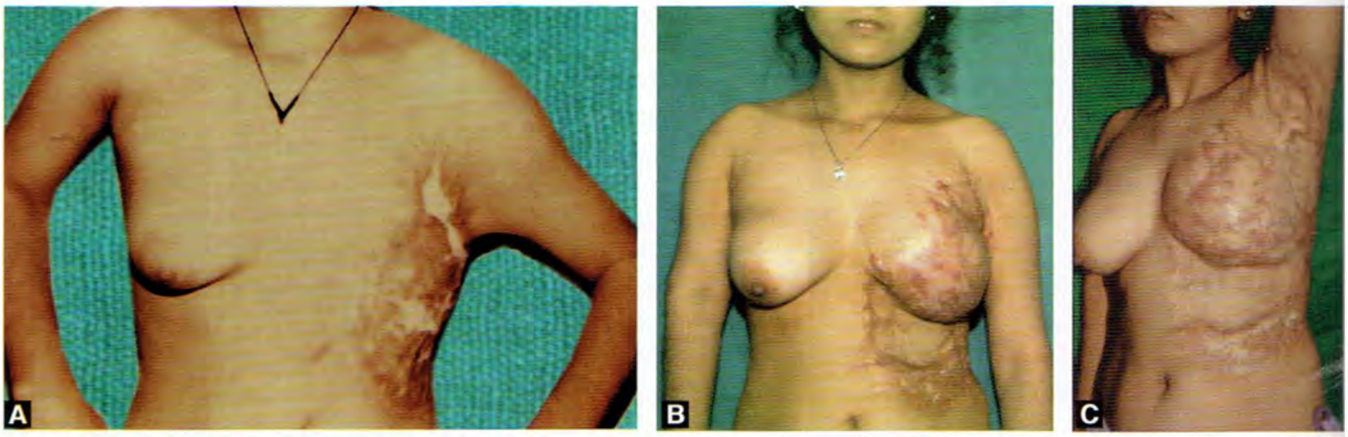


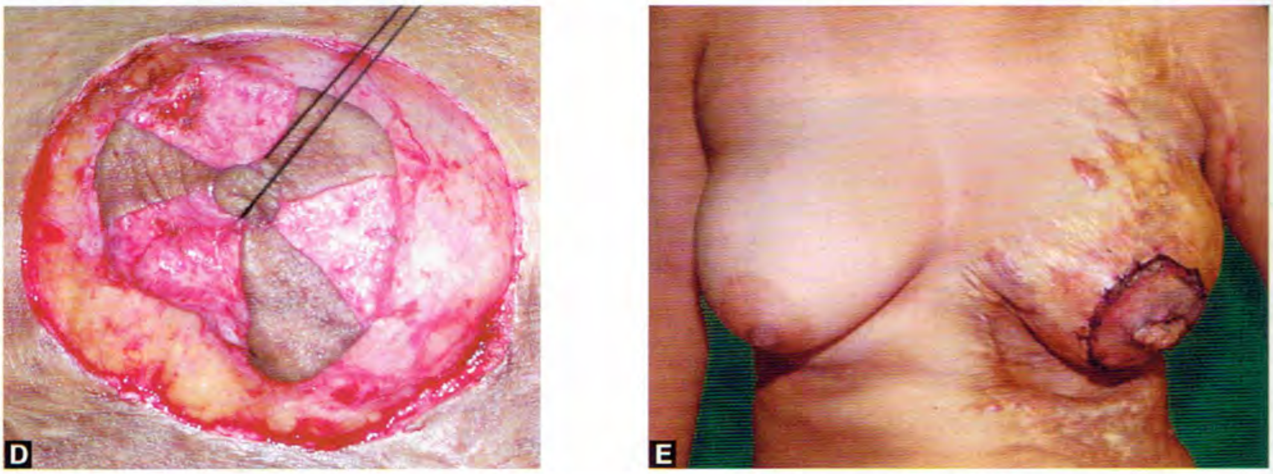
Fig. 34.34B: Postoperative result after release of axilla and skin envelop over the breast covered with split skin grafts. No augmentation was required. Please note the symmetry with respect to size and contours of both the breasts

Release alone versus Excision-release

Except for hypopigmented scars, which are de-epithelialized and covered with thin grafts, scars need not be excised. One should try to release and feed more and more of skin graft. As the breast grows, the graft always falls short in amount and another stage is needed. The hypertrophic scars improve with time and ultimately blend with the grafted areas and other scars due to spontaneously healed burns over the breast. Most of these patients have contiguous scars and/or contractures of the neck, axilla, and abdomen etc. or have these areas split skin grafted during the management of acute burns. Hence, the removal of scars on the breast alone may not be of any significance. In general, it is futile to excise scars and apply grafts as the patient is given good aesthetics in contours only under the clothes. Even excellently taken up and well settled grafts are bad enough for the patient so as not to allow her to wear revealing clothes. Vitiligo skin is a taboo in our Indian society. Moreover, they may be visible through the clothes specially when wet. Hence they need treatment especially in younger



Figs 34.35A to C: (A) A-14-years old girl with contracture of left axilla and apparently absent left breast mound and nipple-areola complex. (B and C) Release and skin grafting were done for axilla and breast envelop. The breast envelop required release twice to accommodate the enlarging breast mound. No augmentation was done. The operated breast appears slightly larger than the normal breast. Front view and lateral view with arm abducted. Postoperative result after four years



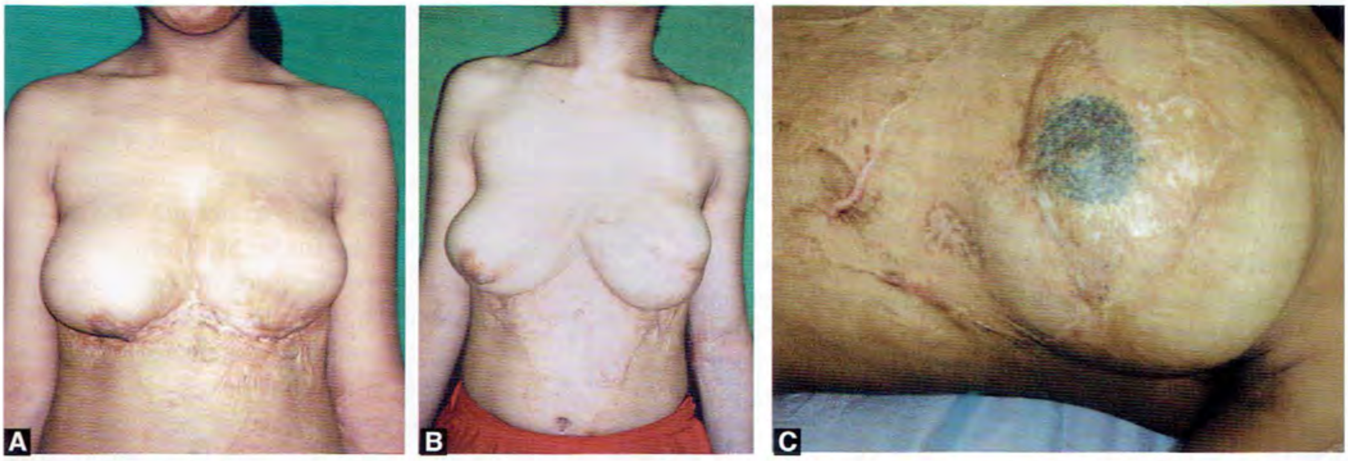
Figs 34.35D and E: (D) Nipple being reconstructed with modified Little's quadrapod technique. (E) Result after one week

age group. When the vitiligo scars are very limited, plenty of donor sites are available and the surgeon is sure that their replacement with a split skin graft will improve the cosmesis, one can excise the scars in continuity with release incisions and graft one larger area with sheet grafts. In patients with extensive vitiligo scars on the anterior trunk, it may serve no purpose by treating those on the breasts only. Once again, the author would like to emphasize that the post burn patient should be treated as a whole and not in bits and pieces.

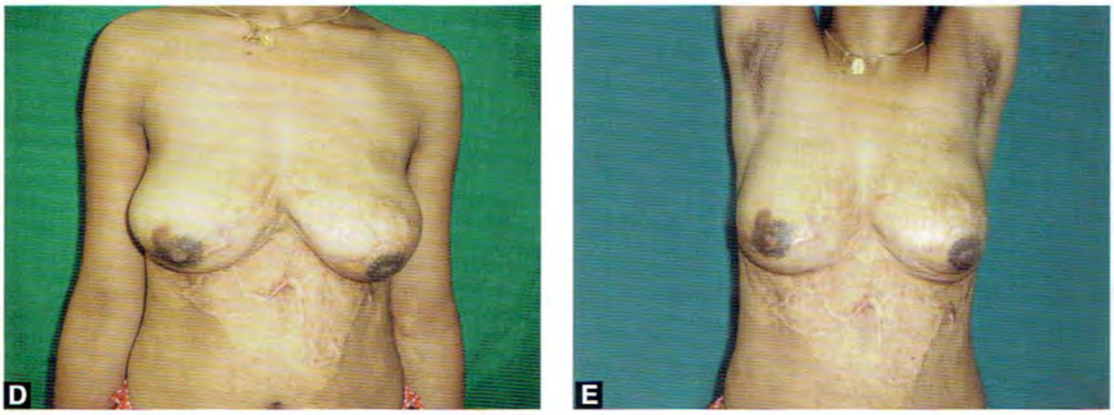
Where to Place the Release Incision

The patient is examined with both arms by the side, then abducted at right angles and finally full abduction with arms parallel and by the side of the head. The position of nipple and areola when present, its position with respect to opposite NAC whether above or below, movement of the mound with arm movements etc. all determine the site of release incision. If the NAC is at proper position it is better to encircle the areola and give long multiple darts in radial directions to release the mound fully.

When release incisions are given, it is of utmost importance that the surgeon does not cut into the mammary gland. This is especially important in incisions made below the areola where the breast fat and the inframammary subcutaneous fat should be identified and the incision deepened between the two. Fortunately the breast tissue (fat) and the subcutaneous tissue over the ribs have different



Figs 34.36A to C: (A) Post-burn contracture of both breasts with loss of left nipple areola complex. (B) Result after release and split skin graft achieving normal contours and infra memory folds. (C) Areola reconstructed by tattooing (darker color has been used as compared to the color of the opposite normal areola as the lighter color is not visible separately from the surrounding skin in our patients



Figs 34.36D and E: Final results with arms by the side and completely abducted, showing symmetrical size and contours of breast and nipple areola complex

color, texture and appearance. Breast fat is lighter in color (Fig. 34.37).

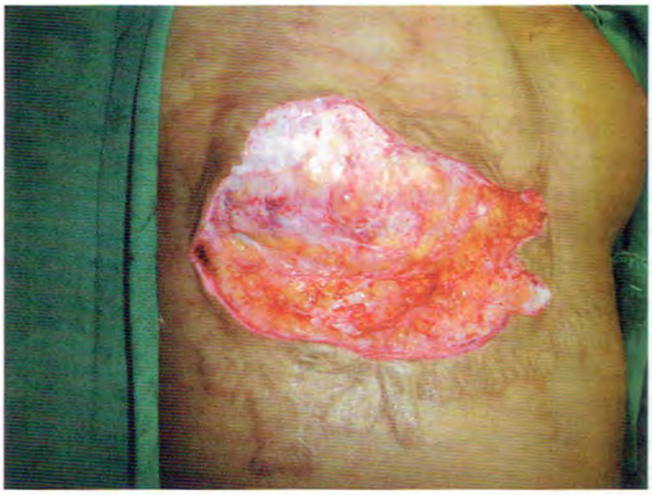


Fig. 34.37: Please note the lighter color and different texture of breast fat in the upper part of the photograph

NAC Reconstruction[48-52]

NAC reconstruction is the final surgical step of breast reconstruction. A large number of techniques have been described and more are being reported. This proves that no technique is totally satisfactory on a long-term basis. Some may not be possible in every case. The biggest problem with all the techniques is deterioration in nipple projection or reduction in the height of the nipple.

Reconstruction of NAC in post burn cases differs from that in post mastectomy cases in many respects. Reconstruction of NAC is an integral part of post burn reconstruction. This is because most of the post burn cases are children and young adults in pre marriage age group with a long life ahead of them. This is in sharp contrast to post mastectomy patients most of whom are middle aged or even elderly and are more concerned about survival and cure from a dreaded disease rather than undergoing another surgery for breast reconstruction. Also in post burn reconstruction, an excellent mound is restored with rather simple and short operations. It is but natural to complete the reconstruction. It is also to be noted that many of the techniques that give excellent results in post mastectomy cases either give poor results or are not at all possible/desirable in post burn reconstruction.

In unilateral cases, the goal of NAC reconstruction is to match the opposite NAC with respect to site, size (projection), shape, color and texture. In bilateral cases, the aim is to restore NAC's which looks similar to each other and to that of a normal female of that age and must be situated at appropriate site. In cases, where the nipple is apparently absent, an effort is made to find it out by pinching the skin at various places, with spine flexed anteriorly and arm adducted, over the probable site and finding out whether the skin is tethered to underlying mound by ducts at any place. If the site of nipple is found, the reconstruction of NAC is done at this site. However if the site is not found, the NAC is reconstructed at a site corresponding to the opposite normal breast in unilateral cases or at appropriate symmetrical site in bilateral cases.

Timing of NAC Reconstruction

NAC should be reconstructed only in a stable mound. If a mound is still increasing in size or changing shape because of normal pubertal changes or because of surgical interventions, the reconstruction of NAC should be deferred till the breast size appears normal for the physique of the patient. An earlier reconstruction can be attempted when the site of NAC on burnt breast is clearly shown by the remnants of the NAC so that the surgeon does not have to select the appropriate site. In cases where the NAC is completely absent and the surgeon needs his own judgment and measurements to find the site, the stability of the mound assumes greater significance. One stage reconstruction of the mound and the NAC is simply not possible in post burn reconstructions.

In post burn reconstructions, if one breast is unburnt, it is the author's firm belief that this breast must not be interfered with for various functional, esthetic and sexual reasons. Moreover, these patients being children and young adults, the normal NAC is never too big to donate.

Reconstruction of Areola

A number of techniques have been described in literature to reconstruct the areola in post mastectomy cases and have been extrapolated to post burn reconstruction. Grafts have been taken from upper medial thigh and labia minora for their darker color as compared to normal skin. These hyperpigmented areas become further pigmented on grafting and may give appropriate color match in post mastectomy cases. However, when the whole breast skin is burnt and there are areas on the breast that have spontaneously healed mixed with grafted areas, the color match of the existing grafts and scars is not much different from these donor areas and they do not stand out. Moreover, in unmarried and adolescent girls even the upper inner thigh skin is of same color as the rest of the thigh from where grafts have earlier been harvested to resurface the post burn raw areas or later to release the contracture of the breast mound. In addition, the darkening of grafted skin, after its 'take', depends on its exposure to ultraviolet light in early postoperative period. This exposure is not there in these patients for obvious reasons. The best technique that is applicable for these patients is tattooing using appropriate colors inserted deep intradermally[53] (Figs 34.36A to E, 34.38A and B). More than one stage is required and any fading at a later date may need re-tattooing to augment the color. No donor site is required and opposite breast is not interfered with.

Reconstruction of Nipple

This is the most difficult and frustrating part of post burn breast reconstruction. It differs greatly from nipple reconstruction in post mastectomy cases. Almost all the techniques of nipple reconstruction have been described primarily for post mastectomy cases and extrapolated directly or with slight modifications to post burn cases. Again, in almost all the described techniques, the reconstructed nipple tends to loose height or projection over a period of time. The availability of a very large number of techniques is a testimony to the fact that none is universally successful.

A number of techniques described for postmastectomy cases are not possible/desirable in post burn cases. For example, nipple sharing technique should not be used as most of these patients are young and their nipple is not large enough to donate. Moreover, one would not like to spoil the only normal nipple in this age group. Composite grafts from the ear lobule or toe pulp are bound to have an unacceptably high failure rate because of the poor vascularity

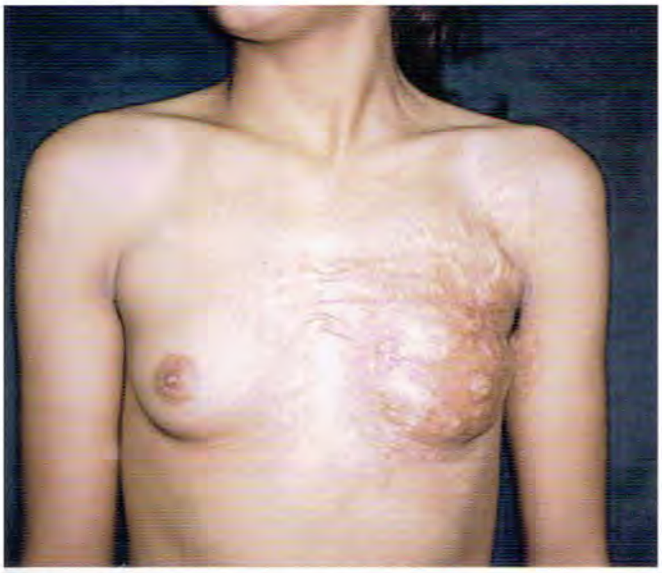


Fig. 34.38A: Post burn loss of left nipple areola complex. Note the reconstructed nipple

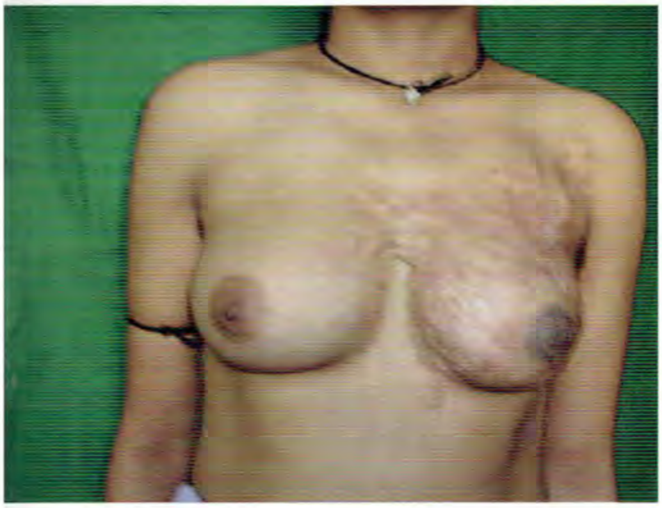


Fig. 34.38B: Tattooing done to reconstruct areola

they get from the skin grafted areas. The techniques where the nipple protrusion is obtained by burying a piece of autologous conchal cartilage or Silastic implant etc. are not desirable because of the risk of jeopardizing the vascularity of overlying split skin graft. Many of the techniques described using local skin flaps to reconstruct nipple projection rely on the dermal and sub dermal base which are highly deficient in skin grafted areas.

The only techniques which are possible are the one which do not raise the local skin (actually skin graft) from its bed so that the vascularity of the skin does not suffer. The author reconstructs the nipple by the following techniques.

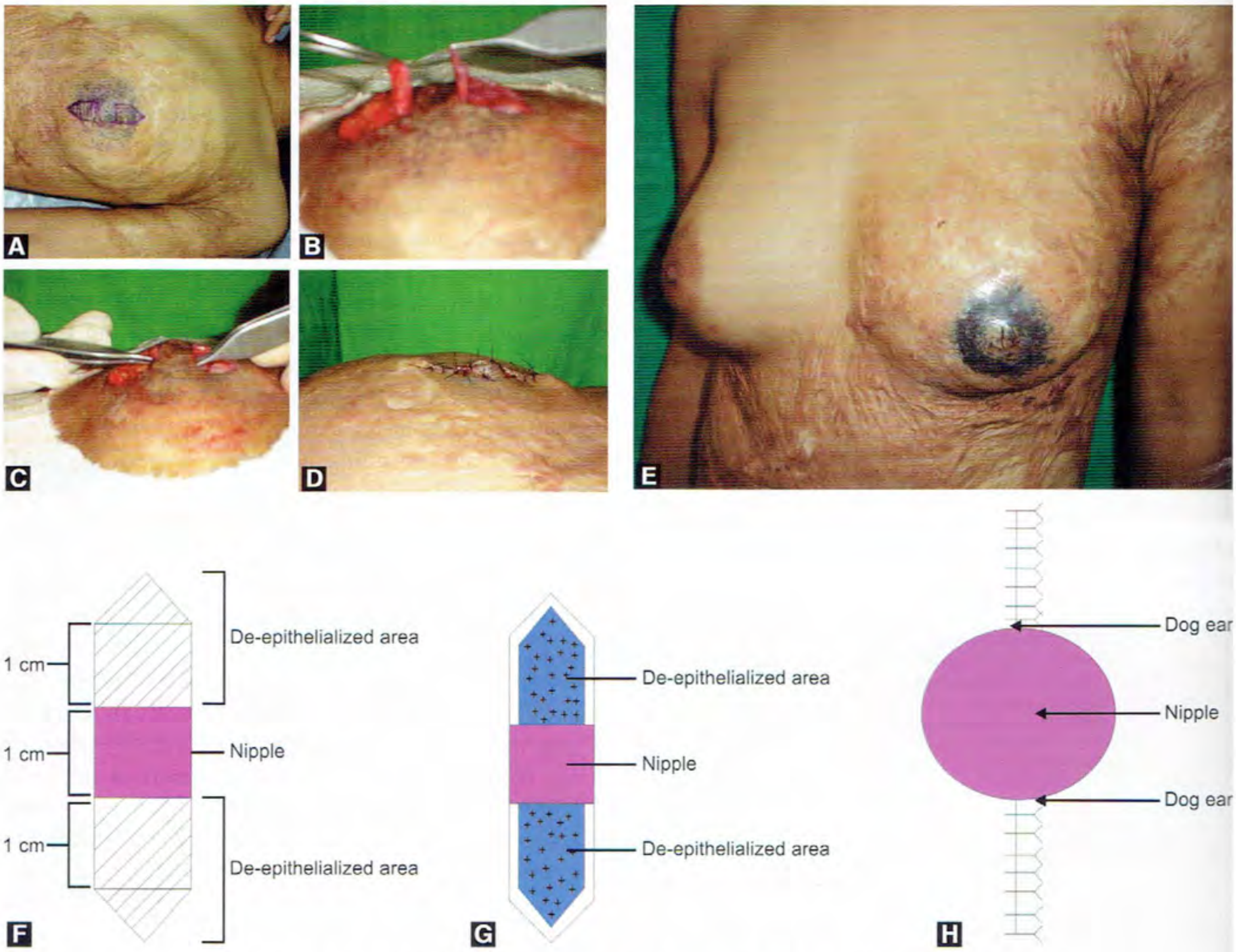
Two concentric circles of 1cm and 4cm diameter are marked with their center at the site where nipple is to be made. Three V-shaped areas are marked in the area between two circles as shown for de-epithelialization. A cut is made in the outer circle and deepened till and slightly into the breast tissue. The de-epithelialized areas are closed by stitching together the adjacent non de-epithelialized skin areas. With a stay suture in the center of the smaller circle, the newly formed nipple is pulled out and the raw area is grafted with thick split skin graft using tie-over sutures. Theoretically, this will give a projection of around 1.5 cm. This over correction takes care of partial flattening over a period of time. After the wounds have healed and the graft settled, size of the areola required is marked with nipple as the center and tattooing done to complete the NAC reconstruction. This technique does not undermine any area and is modified from Little's quadrapod technique[54,55] (Figs 34.35A to E).

Another technique involves construction of two adjacent 'dog-ears' which together give a semblance of a papilla. Three 1cm squares (A, B, B′) are drawn as shown in (Figs 34.39A to H) with square "A" over the site of nipple. Two triangular areas are also marked in the vertical axis as shown. Along with outer two squares B and B', the triangles are thinly de-epithelialized. Two thick dermal flaps are raised with their base towards the nipple, i.e. square "A" and allowed to contract towards the latter. When stitching of the raw areas is started from either apex, a dog ear forms towards the square "A". The dog ears along with dermal flaps help in forming a nipple at site "A".

Male External Genitalia (Penis and Testes)

Although penoscrotal region is very commonly burnt as part of extensive burns, it is highly unlikely for a thermal burn injury to result in loss of penis and/or testis. Contact electrical burn injuries, on the other hand, are known to result in a complete loss of penis and one or both the testes. Their reconstruction is a must not only for restoration of form and function but also for the psychosexual health of the victim. However, one has to wait for the scars in the region to become mature, soft and supple.

The reconstructive techniques for phalloplasty[56-61] have seen great advances since the time of Bogoras who reported the first such operation in 1936. The penis being a tube within a tube and the tube flap having been described by Gillies during World War I, the penile restoration has seen the transformation from multi-staged, random pattern tube flaps to axial pattern musculocutaneous and fasciocutaneous

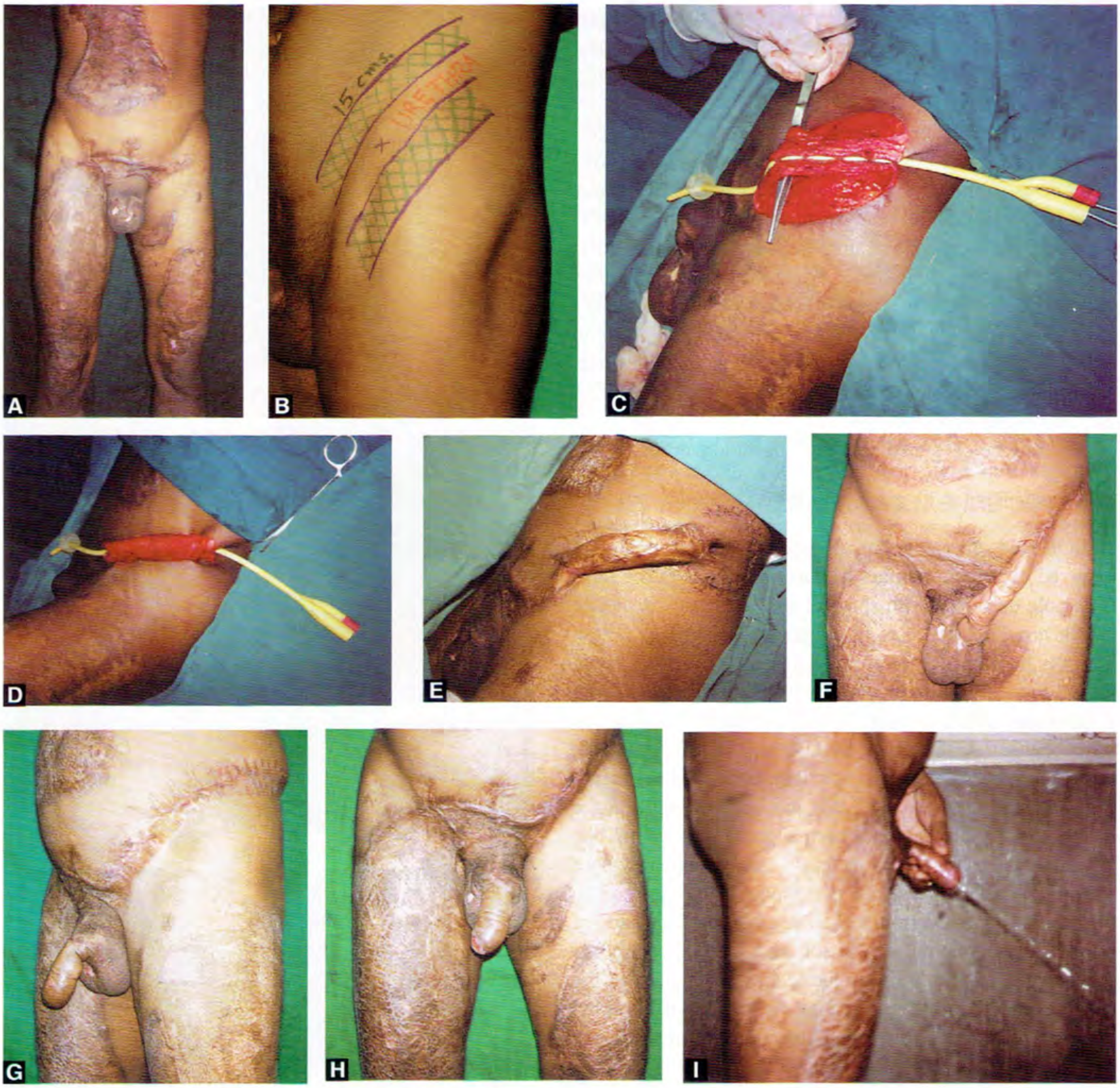


Figs 34.39A to H: (A to E) Nipple reconstruction. Areolar tattooing done (F to H) See text for details

and then free flaps which even included nerves and bone to provide sensation and stiffening. At present, state of the art neurosensory fasciocutaneous free flaps are used to reconstruct a phallus which is acceptable not only functionally and cosmetically but also psychosexually.

For the last 25 years, the free radial artery forearm flap has remained the flap of choice for phalloplasty. The basic design consists of construction of a tube within a tube and various modifications have been done to improve the appearance and sensation and to decrease the complications. The most important drawback of unsightly grafted donor site in the forearm may not be of much concern to a burn patient (compared to patients of gender re-assignment surgery) as he already has plenty of such areas and scars due to initial burn injury itself. However, as the flap is hairy, free ulnar artery forearm flap has been described in its place. The use of fibular osteocutaneous flap for phallic construction has been advocated to achieve rigidity.

The photographs (Figs 34.40A to I) show the staged reconstruction of phallus of a burn victim using a bipedicled, tubed extended groin flap. The urethra has been made from the in-turning of non-hairy skin in the long axis of the center of the flap while the remainder of the flap skin is de-epithelialized. The dimensions of the flap are based on the length of penis desired (15 cm approx.) and width is based on the maximum donor site which can be closed primarily by undermining and advancement with hip flexed. The outer tube is made by stitching the flap margins together over the urethra in such a way that the undersurface of the groin flap forms surface of the outer tube. This necessitates the



Figs 34.40A to I: Post burn loss of penis in a 30-year-old. Reconstruction done in stages by constructing tube in tube from groin region. Patient passing urine normally (Markings on a model)

covering of the raw area with a split skin graft. When the graft and the tube have matured over a period of 3-4 months, division and inset over the region of native penis is started. The use of graft to make the skin of the neophallus lends it some rigidity by virtue of secondary graft contraction.

The silicone testicular implant(s) may be inserted into scrotal skin, if available, or the scrotum is reconstructed from locally available flaps from the upper inner thigh.

Amputations

Electrical contact burn injuries are known to kill the victim instantaneously. The 'fortunate' survivors very commonly end up in multiple and major amputations at various levels leading to a lifelong disability to the victim and a liability on the family and the society. Contact with high tension wires often leads to gangrene of one or more limbs. Circumferential full thickness burns of the extremities with tight eschar, if not treated timely by appropriate escharotomy incisions, may also end up in distal gangrene necessitating amputation(s).

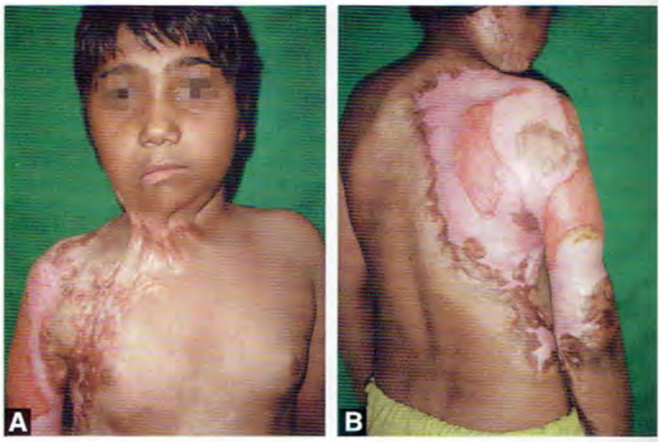
Most of these patients with amputations at higher levels in upper and lower limbs are offered prosthesis to improve function as well as overall appearance. These patients may have stump instability, poor soft tissue coverage over the bone end, joint contracture, atrophic scars, painful neuromas or improper stump length etc. which may pose difficulties in fitting the prosthesis. These problems must be addressed to achieve successful rehabilitation. In upper limb if thumb alone is lost, 'pollicization' of index finger may be done. Microvascular transfer of great or second toe to reconstruct the thumb can give excellent function and cosmesis. Web spaces may be deepened and 'phalangisation' done.

CHRONIC UNHEALED AREAS

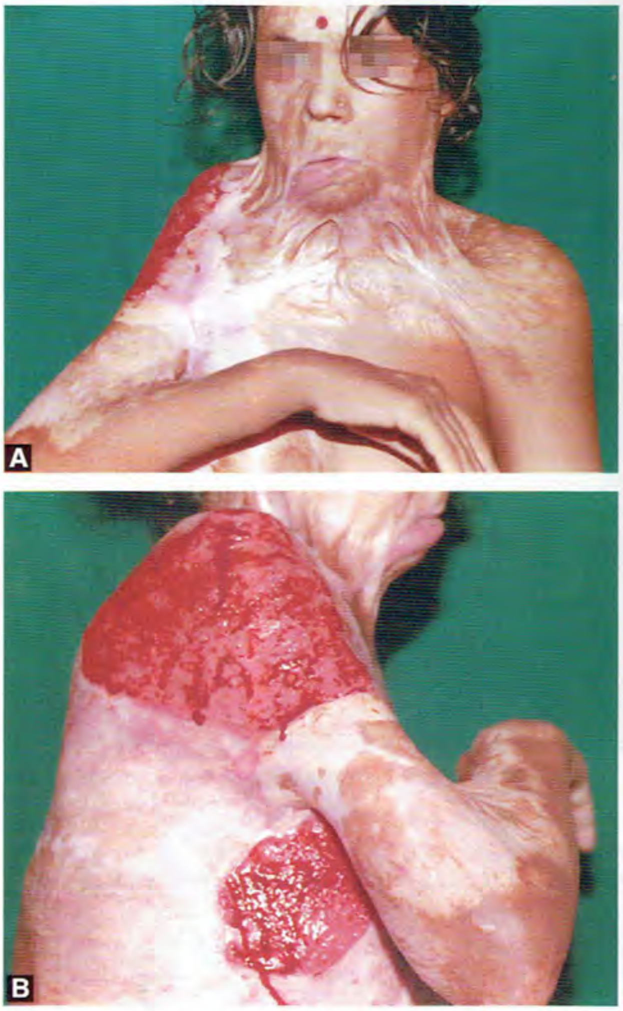
In India and other developing nations, the only treatment received by a large number of burn patients is in the form of simple dressings with some of the commonly available antibacterial creams or even indigenous substances. These patients heal by contracture formation. However, some chronic raw (unhealed) areas may remain over non-joint regions after all the natural limits of the force of contraction have been exhausted (Figs 34.41A to 34.42B). Typically, these areas are over the rib cage, long bones and calvarium etc. Depending on the time that has elapsed between the date of burn and reaching a burn surgeon, the wound bed has more and more of fibrous tissue and consequently poor vascularity. These areas have pale granulation tissue and do not accept split skin grafts well. They may need excision of all the fibrous tissue down to soft and supple healthy bed for a complete graft 'take'. In the scalp region and over the subcutaneous bones, the bed may be formed of the dried up bone which needs chiseling till bleeding bone or diploe in the case of calvarium before wound closure with skin graft or flap.

BONE AND JOINT DEFORMITIES

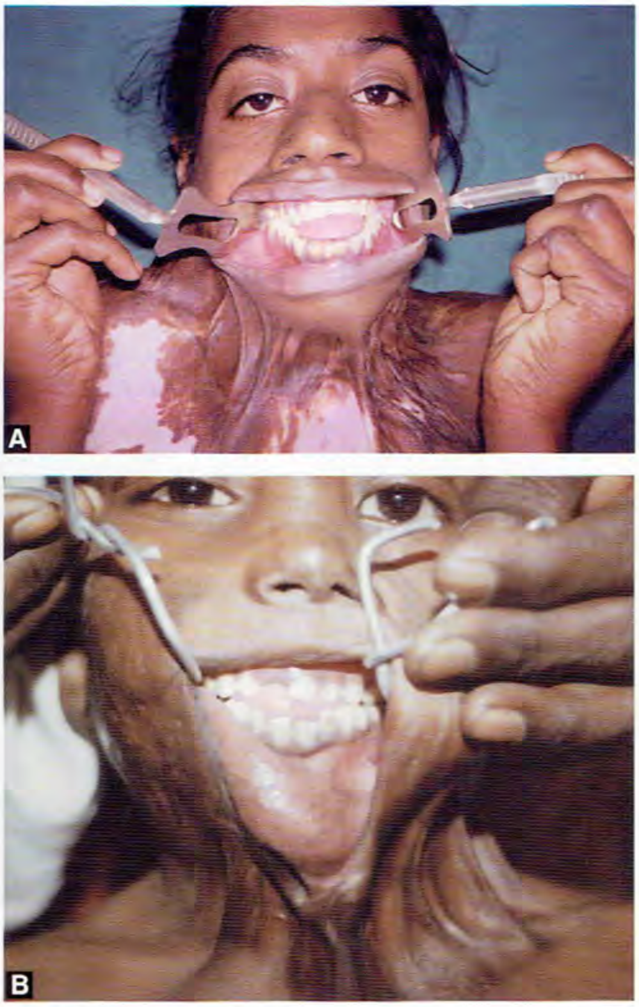
A burn injury may lead to a number of musculoskeletal deformities even if the burn injury was no more than skin deep. Many of these deformities are due to the strong contractile forces which tend to heal a full thickness burn injury in the absence of skin grafting (Figs 34.43A and B). When the burns are sustained in early childhood and not treated appropriately, the deformities get accentuated with



Figs 34.41A and B: Chronic raw areas over lateral arm, shoulder and scapular region along with contracture neck



Figs 42A and B: Chronic raw areas over shoulder, scapula and lateral rib cage where healing by contracture formation is not possible along with post burn contracture of neck, axilla and elbow



Figs 34.43A and B: Deformed mandible with severe malocclusion due to untreated mentosternal contracture following burns sustained in very early childhood

growth of the child. Thus, the joints may be subluxated or dislocated. The bones and joints may bend in an abnormal direction. When such patients come to a plastic surgeon after many years in late teenage or adulthood, it may not be possible to release the joint contracture not only because of shortened musculoskeletal units but also because the growing age has moulded the bone and joint in an abnormal position. Post burn contractures on the dorsum of hand may spoil the extensor apparatus giving rise to deformities like Boutonnière, swan-neck etc. When untreated for a long time, there may be irreversible damage to the joints of hand and permanent life long deformities result.

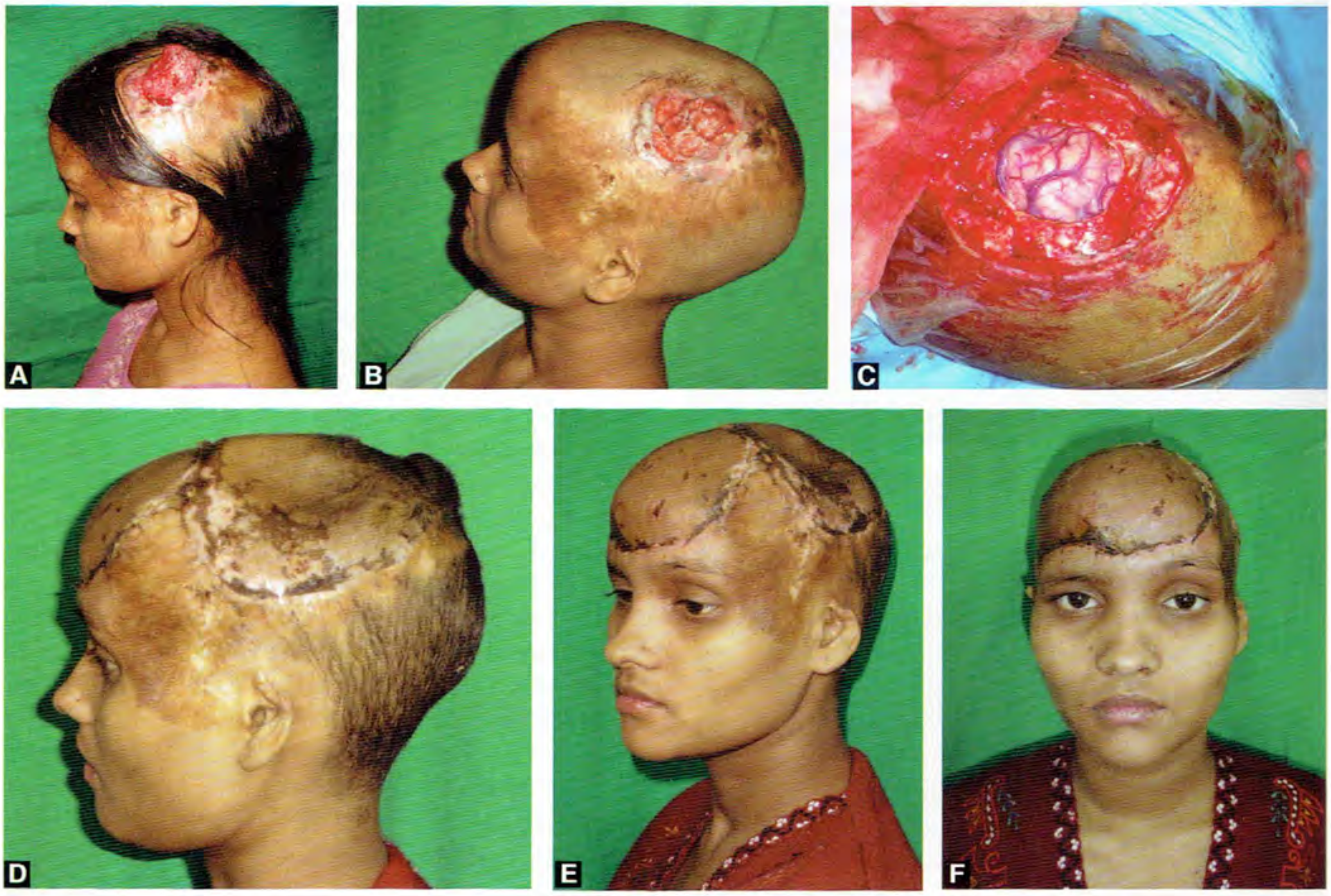
Severe mentosternal contractures in children may lead to deformities of mandible, alveolus and teeth with disturbed occlusion.[62,63]

DEVELOPMENT OF MALIGNANCY

Once the reconstructive surgery of the burn patient is over, all the scars have well settled and matured and the patient has returned back to society, these patients are at risk of development of malignancy even after many decades[64-66] (Figs 34.44A to 34.47E). The fact that thermal burn scars can degenerate and develop into malignancy was known to Celsus in 100 AD. Jean Nicholas Marjolin has been widely acknowledged as to provide with the first description of skin cancers in post burn scars in 1828. However, it was De Costa in 1903 who coined the term "Marjolin's ulcer' and applied it to tumors arising in simple leg ulcers. Some workers have doubted whether Marjolin really recognized the malignant nature of these ulcers. In addition, while some authors restrict the use of the term Marjolin ulcer for malignancy arising out of burn scars only, others use the term for malignant tumors arising in other cutaneous scars, chronic venous ulcers, sinuses of chronic osteomyelitis, pressure sores, scar tissue around colostomies, frost bite etc. as well. In 1930, Treves and Pack[67] presented an excellent analysis on the development of cancer in 34 burned patients. They observed 2% burn scars undergoing a malignant change with extremities and scalp being affected most commonly.

The exact mechanism whereby a malignant change occurs in a post burn scar is still not known. It has been observed that a cutaneous scar subjected to continuous irritation has increased potential for malignant change. Slow initial healing and scar instability are very important in the development of cicatricial cutaneous malignancies. Repeated trauma, when applied to any tight or thickened scar situated over the joints, is an important factor in neoplastic change. The relationship between skin cancer and repeated/prolonged thermal injury is best exemplified by two unique examples of 'Kangri' cancer in India and 'Kairo' cancer in Japan.[68] The 'Kangri' is an earthenware bowl heated by charcoal and worn against the skin of the abdomen and thighs for warmth by Kashmiris in India. In the 'Kairo' burn cancer of Japan, the person keeps a light tin box (with hot coal inside) that fits snugly against abdomen for at least three hours at a stretch to keep warm. Owing to the continued application of heat, the skin in these areas becomes dry and horny and shows chronic dermatitis leading to malignant neoplastic changes.

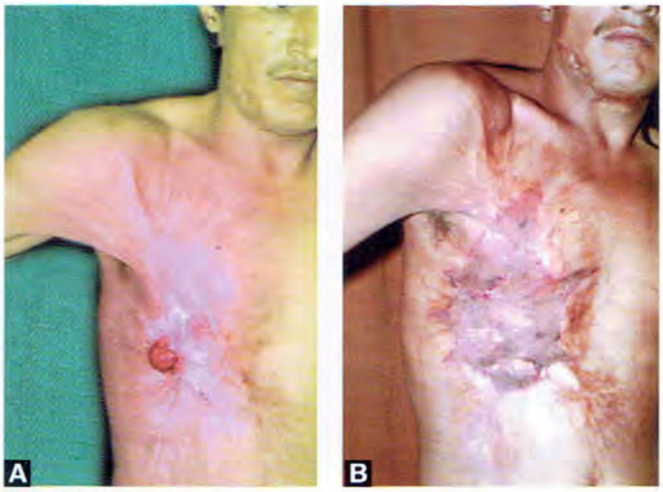
Post burn skin cancers are very slow to grow and metastasize as has been observed and reported by a number of workers. Most patients are free of regional or distant



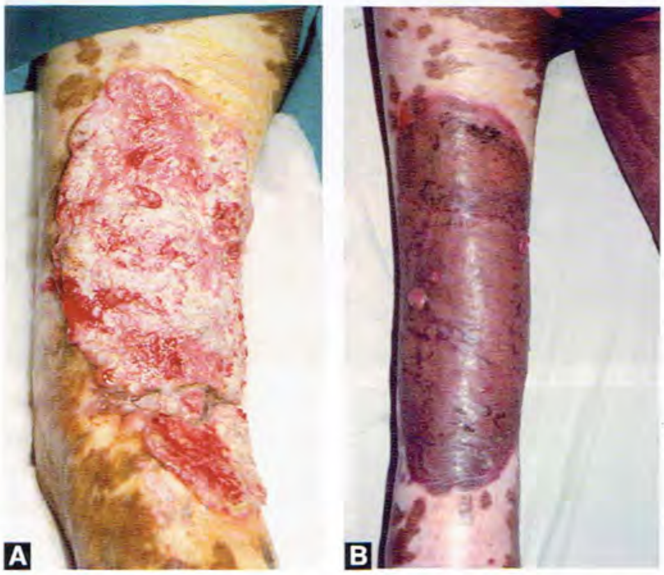
Figs 34.44A to F: 21 year female with squamous cell carcinoma of scalp arising in postburn scars. Treated with wide excision and local transposition of remaining scalp and split skin graft

metastasis at the time of presentation. Once the tumor breaks into the surrounding normal skin or is excised, there is a high incidence of local recurrence and regional/distant metastasis. This has led some workers to suggest the theory (although unproven) of Marjolin's ulcers developing in an 'immunologically privileged site'.[69-71] The presence of relatively avascular dense scar tissue and almost absent/obliterated lymphatics allows the primary tumor to grow unrestricted by host immunological mechanisms. This significant delay may be the reason of the tumor's aggressive behavior once the malignant tumor cells reach the lymph nodes as this is their first encounter with the immune system.

The most common malignant neoplasm to develop in post burn scars is the squamous cell carcinoma (SCC) with basal cell carcinoma(BCC) being the next. Of the 1091SCC's studied by Treves and Pack, 21(1.92%) arose in post burn scars. Seven BCC's arose in burn scars out of 1374 cases studied (0.51%). There are a number of case reports of various other cancers including sarcomas developing in post burn scars.[72] Thus, melanoma, adenocarcinoma, liposarcoma, leiomyosarcoma, fibrosarcoma, malignant fibrous histiocytoma, even extraskeletal osteogenic carcinoma, malignant schwannoma, etc. have all been reported. The relatively deep position of mesenchymal cells of the dermis and subcutaneous tissues making them less vulnerable to trauma and their lesser tissue regeneration than the superficial epidermis has been put forward as the explanation for rarer occurrence of sarcoma as compared to carcinoma in burn scars. Marjolin's ulcer is three times more common in men as compared to women. Roughly, it involves lower limbs, head and neck, upper limbs and trunk in a ratio of 4:3:2:1. All post burn cicatricial malignancies develop with a prolonged latent period, i.e., the time interval between the acute burn injury and the onset of malignancy. This period has been reported to vary from 8 to 63 years with a mean of 32.5 years by Treves and Pack (1930).



Figs 34.45A and B: 38 years old male with squamous cell carcinoma over anterior ribs arising in post-burn scars treated with wide excision and split skin graft



Figs 34.46A and B: (A) Squamous cell carcinoma arising out of post burn scar in popliteal fossa region. (B) Wide excision and split skin grafting done. Result after two months

Rarely, early arising Marjolin ulcer cases have been reported with a latent period of one year or less.[73] In addition, the latent period has been found to be inversely proportional to the patient's age at the time of burn, i.e. the younger the victim at the time of burn, longer is the interval for malignant change. Older the patient, shorter is the latent period.

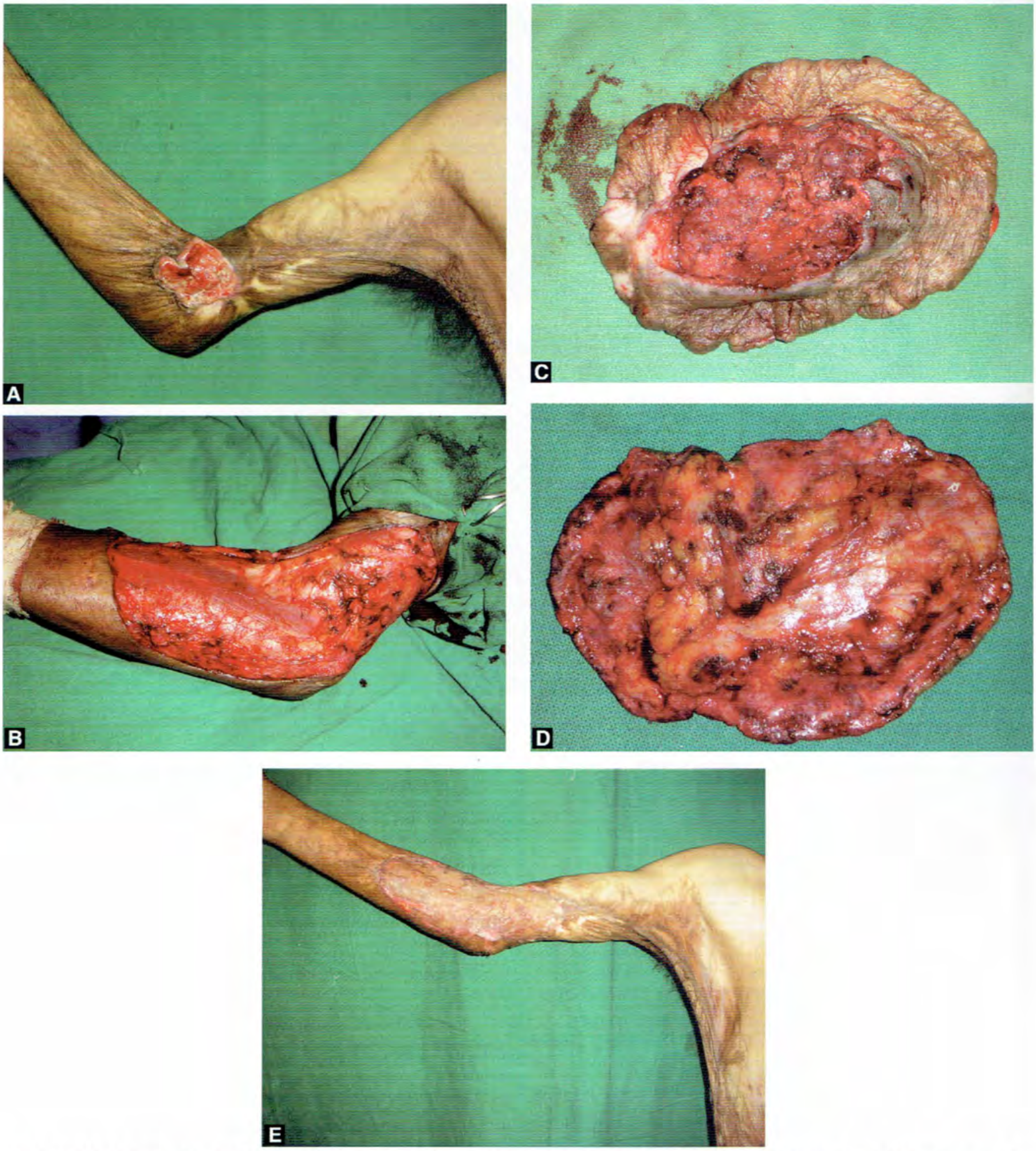
The post burn scar malignancies are to a great extent preventable. Indeed, their incidence has certainly decreased. This is because of the early treatment of unstable and tight scars and ulcers especially over the joints where repeated trauma occurs. The grafted as well as spontaneously healed burnt areas must be kept soft and supple by application of bland oils and creams. Once the acute as well as reconstructive treatment is over, all patients must be advised to report any ulceration at the earliest. Any patient presenting with a non-healing ulcer in an earlier healed burn area must be looked upon with suspicion and subjected to excision biopsy. The contracting bands must be released and the area split skin grafted.

Treatment of biopsy proved malignancy depends on the histopathological type and the status of lymph nodes draining the area. For squamous cell and basal cell carcinomas, a wide local excision with a 3-4 cm margin of normal skin and including the muscle fascia should be done. Although some workers have done prophylactic lymph node dissection, most recommend this surgery only when they are palpable. It is to be noted that the enlarged lymph nodes palpable at the time of initial presentation may be due to chronic infection. Fine needle aspiration cytology (FNAC) examination may be done after a course of broad spectrum oral antibiotics. The nodes tend to settle after the local wound heals in 2-4 weeks time. A regular life-long follow up is essential because metastatic recurrences are common within a 3-year period. Squamous cell carcinomas arising in burn scars have always been considered as more aggressive than arising otherwise. Poor prognostic features are lymph nodal involvement and local extension of disease at the time of presentation. Involvement of the regional lymph nodes is followed by metastasis to brain, liver, kidney and lungs. Lower extremity lesions have higher rates of nodal metastasis. A patient who is metastasis free for three years has an excellent prognosis. Malignant lesions which involve deeper structures like bone and joint are treated by amputations at appropriate levels.

The management of the rare sarcomas in burn scars is no different from those which arise without any such history of burn or other scar.

MISCELLANEOUS

It is said that a major burn is a systemic disease and affects the physiology of each and every system in the body. It is not surprising, therefore, that the list of post burn sequelae is almost unending and cannot be classified adequately. Each and every sequela described in the vast burn literature cannot be covered even in a book devoted to same. A number of problems must, therefore, have been left out in this chapter.



Figs 34.47A to E: (A) Squamous cell carcinoma arising in a post burn scar at right elbow region with contracture. (B) Wide excision and release done. (C and D) Views of the resected specimen. (E) Postoperative result

For example, electrical burns are known to lead to cerebral, spinal and peripheral nerve injuries with a long latent interval between the injury and the neurological dysfunction or deficit. They may cause hemiplegia, quadriplegia, paraplegia and neuropathies. These patients need the consultation of a specialist neurologist or neurosurgeon. Cataract can also occur requiring an ophthalmologist's intervention. Heterotopic ossification or myositis ossificans is a rarely encountered complication of burns. Although elbow is the most common region involved, shoulder, hip and knee may also be affected and require surgical treatment by an orthopedic surgeon. Respiratory and inhalational burn injuries may lead to long term lower respiratory tract instability and need management by a respiratory physician.

PSYCHOLOGICAL DISORDERS

Beyond physical sequelae, a burn injury can also result in severe psychological and emotional distress due to the traumatic event itself and due to the scarring and deformities left even after the exhaustion of a plastic surgeon's armamentarium. It is said,"Time is a great healer". A burn surgeon realizes the truth of this statement not only in the healing of even extensive wounds but also in the recovery from emotional trauma. However, scars whether physical or psychological do remain to a greater or lesser extent. In contrast to other injuries and adult victims, the burn injuries in children do cause psychological problems in parents as well. This is because of their concern for the child's future, especially with respect to finding a suitable match, when they grow up. Not only for victims who have post burn sequelae on the face is it difficult to find a match but also for patients who have scars in usually covered areas especially on the breasts as this fact cannot be disclosed to all prospective life partners. Parents of girls in marriageable age group do move from one plastic surgeon to another in the hope that some 'cosmetic surgery operation' may solve their problem or at least there must be some cream or lotion the application of which will fade away the scars. The large number of proprietary creams available in the market for the purpose and their high turnover in amount of cream and money is a testimony to the fact that the patient and their parents are ready to do anything to achieve scar less skin. Expression of inability to do any further by the plastic surgeon leads to depression.

SUMMARY

The burn is a devastating injury with a lot of immediate morbidity and mortality to the victim, great financial implications to the patient, his/her family and to the government and an unending list of post burn sequelae and the ever changing improvements in techniques to manage them. Full textbooks have been devoted to the management of post burn sequelae [74, 75] apart from the vast literature available. This chapter attempts to present the commonest problems and principles of their management over which the reader can build up from his own experience and study of further literature.

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Chapter

35

Medicolegal Aspects of Burn Injuries

Chandrakant, Sujata Sarabahi

INTRODUCTION

Fire has played a multipurpose role in the Indian history. It has been used not only for cooking food but also for religious purposes (like 'havans' and 'sati'), blessing couples during marriage ceremonies and also for sterilizing instruments for surgery (Sushruta). Over the years, it has been very often misused for committing homicide or suicide or even for destruction of evidence of the crime committed. Even though 'dowry deaths' due to causes other than burns were prevalent in the West in the middle ages, 'bride burning' or 'dowry deaths' has continued to plague the Indian society even in the twenty first century. Thus, burns in any female within seven years of marriage are considered significant and culpable before the law until proved otherwise.

The Webster's dictionary of English language has described **'Burns'** as the injuries caused by application of dry heat, moist heat, abnormal cold, chemicals substances, poison, electricity or lightening and characterized by reddening, blistering or charring. For medicolegal purposes, injuries caused by lightening, electricity, X-rays, radiation, friction of body tissues, ultraviolet radiation are also included into burn injuries.

Scalds are a kind of burn injuries resulting due to the application of liquid object, usually hot water and other liquids at or near their boiling point or from steam. There is no singeing of hair or charring of tissues. It resembles like a first degree burns showing reddening with formation of blisters with different shape and size, with well defined, demarcated line of differentiation. If punctured, there is a red base.

Chemical burns are caused by corrosive substances like acids or alkalies, characterized by redness of skin, formation of ulcers, acute inflammation and discoloration and staining of skin.

CLASSIFICATION OF BURNS

There are four types of classifications usually used for burns.

Dupuytren's Classification

Dupuytren has divided the burn injuries into six degrees depending upon the loss of external and internal tissues of the body.

1st Degree Burns: There is simple redness of skin, erythema and after healing there is no scar formation.

2nd Degree Burns: There is acute inflammation and also formation of blisters. After healing, there is no scar formation. Burns are very painful.

3rd Degree Burns: There is destruction of cuticle and part of true skin and subsequently healing results in formation of scars.

4th Degree Burns: There is loss of whole skin with loss of nerve endings. After healing, there is formation of scars. But burns are not painful.

5th Degree Burns: There is loss of deep fascia, internal tissues and at the time of healing there is formation of scars, associated with deformities.

6th Degree Burns: There is charring of external and internal tissues with injuries to whole limbs including bones and other organs of the body.

This classification is not accepted any longer because it is difficult to differentiate the various degrees by naked eye and they do not relate well to the prognosis.

Wilson's Classification (Modern Classification)

Wilsons classification is also known as modern classification where Dupuytren's classification has been grouped into three types of nomenclature:

i. **Epidermal (1st and 2nd Degree Burns):** The burned part is red and there is formation of blister which is covered by pale, avascular epidermis surrounded by a thin bright area of inflammation, but on healing no scar formation results.

ii. **Dermoepidermal (3rd and 4th Degree Burns):** There is loss of cuticle and part of true skin (whole thickness). On complete healing, there is formation of scars. These types of burns are very painful due to the irritation of nerve endings surrounding the affected tissues.

iii. **Deep (5th and 6th Degree Burns):** There is charring of external and internal tissues including bones with destruction of nerve endings. Burns are relatively less painful. Healing results in formation of scars, contractures and deformities.

Rule of Nine Classification

Surgical classification also known as Hebras classification or Rule of Nine—depending upon the estimation of percentage of body surface burnt. The whole body is taken as 100% and each part of the body is given 9% or multiple of 9% as follows:

Part of Body	*% of Body Surface*
Head and neck	9%
Right upper limb	9%
Left upper limb	9%
Right lower limb	18%
Left lower limb	18%
Anterior trunk	18%
Posterior trunk	18%
Perineum	1%
Total	100%

Clinical Classification for Medicolegal Purposes

According to this classification, depending upon the destruction or damage to epidermis or dermis, burns are grouped into two categories:

i. **Partial thickness burns:** When there is loss of epidermis and part of dermis. These burns are very painful.

ii. **Full thickness burns:** When there is loss of whole of epidermis and dermis. These burns are painless.

EFFECTS OF BURNS

Effects of burns mainly depends on the following factors:

a. **Duration of exposure:** Greater the duration of exposure more severe are the burns

b. **Age:** Young children, infants and older individuals suffer from burns more severely than adults

c. **Temperature:** Higher the temperature greater the damage and severe the burns as the human skin is tolerant to a very narrow range of external temperatures, usually between 20°C and 44°C. Lower temperatures which can be tolerated for short periods can cause burns if exposure is prolonged. Similarly an object at 44°C can cause burns if applied for five hours or more. On the other hand, at 60°C, only 10 seconds exposure is sufficient to cause a burn injury. 'Kangri burns' are very commonly seen in Kashmiris due to the close contact of lower abdomen with cisterns called "kangris" for the purpose of warmth.

d. **Extent and Part of Body:** Surface area and part of the body are more important than the degree of burns. Burns on the head, neck, trunk and genitals are relatively more dangerous than on other parts of body.

OBJECTS PRODUCING BURNS

Object producing burns are: (a) Highly heated solid objects or molten metal, (b) Flames causing singeing of hairs, blackening of skin, (c) kerosene oil, petrol, etc. produce sooty blackening, (d) Explosives produce tattooing and blackening, (e) X-ray and radiation causes redness of the skin with shedding of hairs and pigmentation of the surrounding skin, (f) Ultraviolet rays causes acute eczematous dermatitis and (g) Corrosive substances cause ulcerated patches.

For a forensic expert causative agent is required to be known after death, so that based on the details of the appearance the causative agent can be identified with certainty.

The commonest burns all over the world are the ones sustained due to dry heat which cause damage due to conduction or radiation. If applied beyond a certain time, they cause charring or coagulative destruction in the body. Burns from flames usually leave shadow areas of unaffected skin like in the creases (axilla, groin), which are spared from direct exposure.

If exposure to flames is for very short duration, only the tips of the hair all over the body are singed (eyelashes, eyebrows, pubic and scalp hair). The melting of the keratin in the distal end of the hair and its resolidification on cooling leaves a blob on the end of the hair shaft. If the duration of exposure is prolonged, there is complete burning of the hair.

Corrosive burns: Corrosive burns are rarely accidental. Usually they are associated with a homicidal intent, as seen in the Indian scenario. Death due to corrosive burns is uncommon and homicidal attacks are done with the intention to maim the victim and not to kill. The most common agent used is sulfuric acid which is also called 'Oil of Vitriol' and the act is, therefore, called "Vitriolage".

The burns due to acids and alkalis usually give a trickle-like pattern. The nature of injury produced in the skin is usually typical for different corrosive agents and hence the appearance of the wounds is an indication for the nature of the causative agent. Sulfuric acid-destroyed areas show a grey or black appearance; nitric acid produces yellow brown discoloration of the skin. Strong alkalis react with skin and tissue lipids to give the wounds a soft, slippery and slimy look which is initially grayish and later on attain a brown tinge. Prolonged exposure to petrochemicals like kerosene and petrol results in blistering. The blisters are initially creamish and turn yellow after a short time.

If burns are due to splashing of hot liquids, a trickle pattern is seen which indicates the posture of the patient at the time of contact. The worst scalds are seen in the area of initial contact and it decreases in intensity as the cooling liquid flows away. Whereas, in case burns are due to immersion in hot liquids, the severity of damage is uniform till the sharply demarcated margin. Scalds do not cause singeing of surface hair as in case of burns due to dry heat.

THE BURN INDEX

This index system was developed by the **Brook Army Medical Centre**, Burn Unit of the US Army, for ascertaining the effectiveness of treatment and prognosis of burns. According to this system 0.5 point is denoted for each percent area of second degree and 1 point for each percent of third degree burns. At about 45 points, the mortality rate is calculated to be approximately as 50%.

ANTEMORTEM AND POSTMORTEM BURNS (FIGS 35.1 TO 35.4)

Many times, burnt or charred bodies are discovered and the question arises whether the victim succumbed to burns or otherwise. He or she may have died due to inhalation of toxic fumes or due to some other reason like strangulation or other violent acts and then burned later. It is, therefore, essential to differentiate the antemortem burns from postmortem burns by their characteristic features (Table 35.1)

Postmortem heating of body may mimic the antemortem appearance. Postmortem drying of weeping serum in a burnt area may lead to parchment like surface which can be mistaken for third degree burn. If the skin actually catches fire black brittle masses of dead burnt skin are formed under which the muscles also may show a cooked appearance. Muscle contractures from prolonged heat exposure are always a postmortem finding since cooking of muscle is incompatible with life. This is more commonly seen in the flexor muscles which produces a 'boxer's 'or pugilistic attitude. On the other hand, contracture of paraspinal muscles produces opisthotonus.

Postmortem electric burn marks appear similar to antemortem electric burn marks except that they lack the vital flare and erythematous changes. Postmortem electric burns may be so severe to give a cooked muscle appearance.

Besides these, there are certain postmortem artifacts seen in burn cases which are as follows; *Postmortem external wounds*: Skin, if heated after death, contracts markedly leading to splits in the body surface called 'heat ruptures' which mimic antemortem wounds and are seen on the extensor and flexor surface of the joints. Though these may be seen following forceful attempt to straighten a limb after death. They can, however, be differentiated from the antemortem wounds by absence of bleeding in the deeper tissues but it may be difficult if the burns are very deep and involve the deeper tissues also.

Heat hematoma: This is found following prolonged application of intense heat to the head which causes the brain to compress into a cooked mass, the dura may split and blood may boil out of the diploe or venous sinuses as a chocolate brown spongy mass. At times, even the brain may ooze out as a frothy paste. The skin as well as underlying skull bones may be totally charred. These changes have to be differentiated from antemortem skull fractures.

Heat fractures: On postmortem, some charred bodies often show skull fracture and intracranial hemorrhages. These arise either due to violence before death or action of heat on skull and brain or injury to head after death. Antemortem fractures can be differentiated from postmortem fractures by the presence of tissue reaction at the margin of the fractured bone on microscopy, which would occur only if the victim survived for at least an hour after injury. In case victim dies in lesser time these changes will not be seen, instead there will only be external signs of trauma.

Table 35.1: Differentiating features of antemortem and postmortem burns

S.No.	*Antemortem Burns*	*Postmortem Burns*
1. Evidence of vital reaction, line of redness.	Present	Absent
2. Vesicles	Fluids present containing albumins, chlorides	No fluid-only air present
3. Inflammation	Signs of inflammation, formation of granulation tissues, pus and sloughs.	Absent
4. Healing	Process of reparative changes present	Absent
5. Enzymes Estimation	Increase in Enzymes Estimation	Nil
6. Carboxy Hemoglobin in blood	Present	Absent
7. Carbon Soots in upper respiratory tracks	Present	Absent
8. Pugilistic Attitude/Boxer Attitude	Present	Absent
9. Carbon soot in stomach due to reflex swallowing	Present	Absent

Heat fractures of skull have been described as two types by Mortiz:

a. Increased intracranial pressure forcing contents of skull outwards leading to an explosive fracture. These fractures are seen along suture lines along with brain matter exploding out in a circular pattern.
b. Drying and shrinkage of outer table leading to stellate fractures and fragmentation of outer table which cracks on handling, during postmortem, thus mimicking a depressed fracture. However, in antemortem depressed fractures, the outer table shows shelving along the edges of the fracture.

CAUSES OF DEATH IN BURNS

The death in burn injuries may occur from:

1. Primary Shock/Primary Neurogenic Shock due to pain, fear or inhibition
2. Secondary Shock due to loss of fluids is the most common cause of death in first 24 hours.
3. Asphyxia or due to inhalation of smoke, carbon dioxide, carbon monoxide gases.
4. Toxemia
5. Septicemia—when patient survives for some duration, is the most common cause of death in the later period.
6. Acute Renal Failure with associated Bio-chemical disturbances
7. Gastrointestinal Disturbances including Curling's ulcers
8. Other complications, like tetanus, gangrene, amyloidosis etc.

Spontaneous combustion of the human body cannot occur as the human body is composed of seventy five percent of its own weight of water.

Preternatural combustibility is rare. Inflammable gases like Hydrogen sulfide, methane or Hydrogen are sometimes formed in the alimentary tract due to bacterial fermentation during digestive process. These gases are belched out from the anal region and if a flame or flame producing object is nearby the body, it may cause burns at the site. Similarly during surgical procedures on large bowel or near that area, while using electric cautery a mild explosion may occur due to ignition of these gases. However, complete combustion of body is impossible.

MEDICOLEGAL ASSESSMENT OF A BURN VICTIM

A Medical Officer, Forensic expert, Forensic Pathologist or any other doctor, who is authorized by the concerned State Government by issuing a Gazette Notification under sub-section 3 of Section 174 of the Criminal Procedure Code can conduct Medicolegal autopsies. Basically, the following facts are required to be determined in case of alleged deaths due to burn injuries (1) Identification of the deceased—age, sex, height (2) whether the burn injuries are antemortem or postmortem in nature (3) what are the causes of death in the deceased and (4) whether the burns are accidental, suicidal or homicidal.

Identification

In majority of the cases of burns, bodies are already identified prior to Medicolegal autopsy. But in cases of unidentified bodies, detail examination of bones, jaws, pelvic bones, status of teeth, obtaining finger prints from less damaged hands or feet need to be done. In case of charred bodies, marks of external wounds may be destroyed and determination of even age and sex may be difficult. However, even at 2000° F temperature all body parts are not destroyed. Fragments of remaining long, short bones, pelvic bones, teeth and jaw are carefully examined and X-rays are evaluated. A detailed DNA finger printing further helps in the identification.

Accidental Burns (Fig. 35.1)

Accidental deaths due to burns are generally reported at the place of work like kitchen, factories, in burning houses or buildings or shops, Cinema Halls (like in case of Uphaar Picture Hall Tragedy, New Delhi), fire in some exhibition (Shastri Park, Meerut, Uttar Pradesh), hotels (Siddhartha Hotel now known as Vasant Continental Hotel, New Delhi) and rarely in train compartments. Large number of deaths in accidental burn injuries also occurs among aged persons, infants, children, intoxicated or drugged persons, or persons suffering from epilepsy or sudden syncope. Mainly the women and men working in the kitchen, sustain burn injuries due to leakage of cooking gas, bursting of kerosene oil stoves (Fig. 35.3C) which is very common in India, falling of kerosene oil lamps, burning candles during sleep, warming during winter seasons from electric heaters or wooden fire in remote areas of smaller cities and villages.

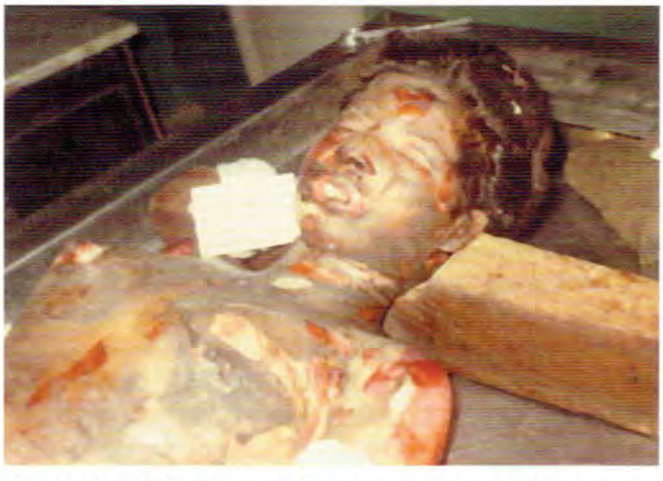

Fig. 35.1: Case of accidental 100% antemortem burns showing singeing of scalp hair, blackening of face, neck and upper chest due to clothes catching fire following bursting of kerosene stove

Suicidal Burns (Figs 35.2A to 35.3C)

Suicidal burns are relatively more common amongst Indian women than males due to increasing tension in day-to-day life with domestic problems, harassment by husband or his family members, financial crisis, chronic illness, loss of life of near and dear family member, depression or suicidal tendencies. Careful evaluations of circumstances along with detailed postmortem examination findings are evidence to suggest suicidal burns. Self immolation is also a way of public protest against some organization or Government policies, in which the protestors, in public or other places which are in public view pour kerosene oil on self and set the body on fire. In suicidal burns usually some inflammable material like kerosene oil, Petrol or Alcohol is used. The material is poured

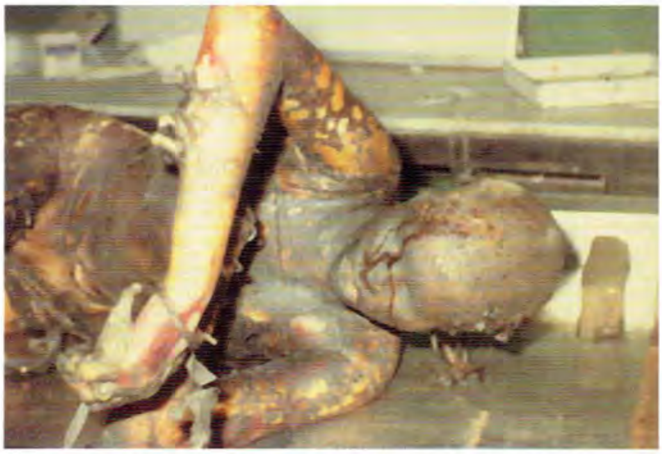

Fig. 35.2A: 100% suicidal antemortem burns showing burned scalp hair and charring of both upper arms

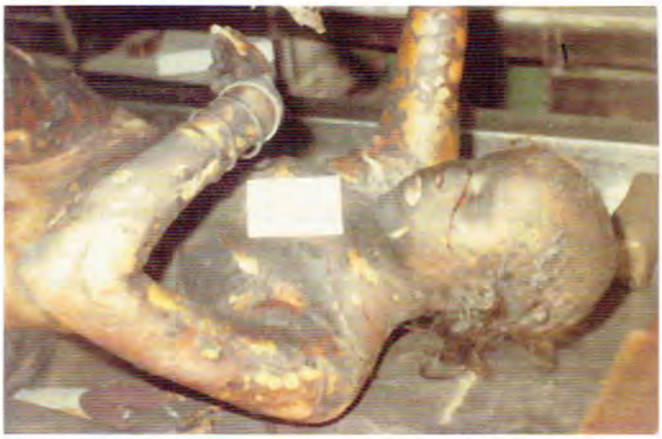

Fig. 35.2B: Same case in pugilistic attitude with heat rupture in right inguinal region. Intact bangles indicating no signs of struggle

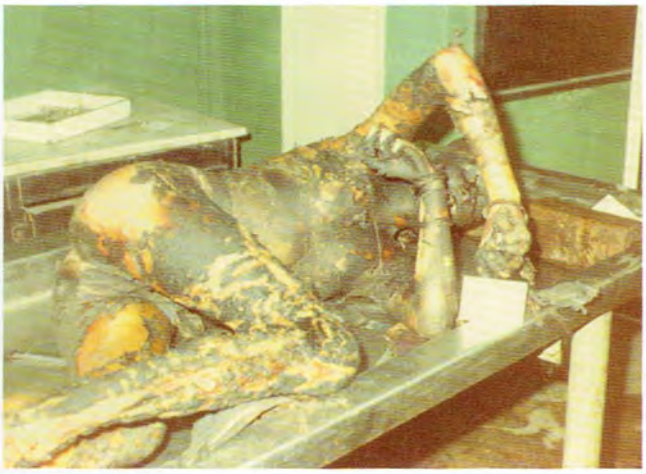

Fig. 35.2C: Same case with pugilistic attitude, charring of skin of right forearm, chest, lower thigh and knee with heat ruptures in left lower leg

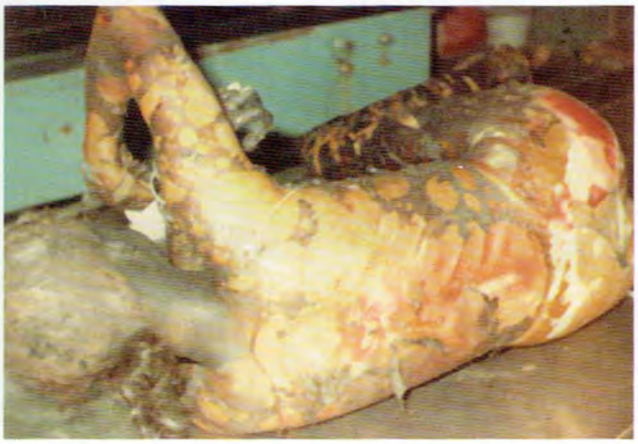

Fig. 35.2D: Same case with charring of skin of scalp and tuft of scalp hair with area of redness over right hip

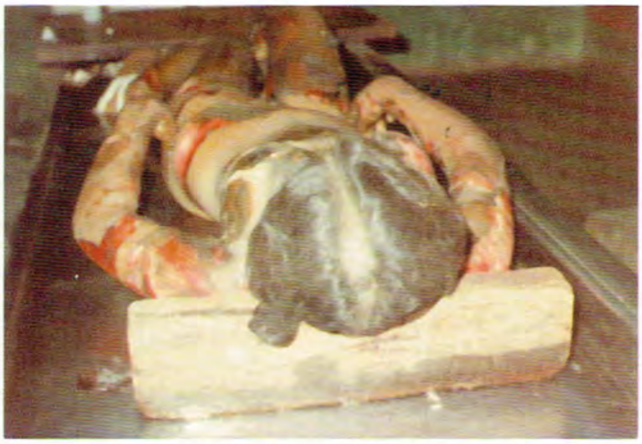

Fig. 35.3A: Suicidal burns showing burning of scalp hair and scalp skin in the middle

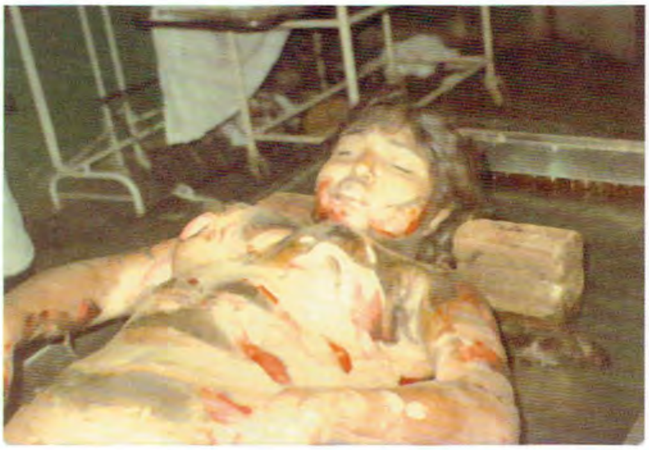

Fig. 35.3B: Same case with singeing of scalp hair anteriorly indicating suicide by pouring of kerosene oil on head also before igniting it

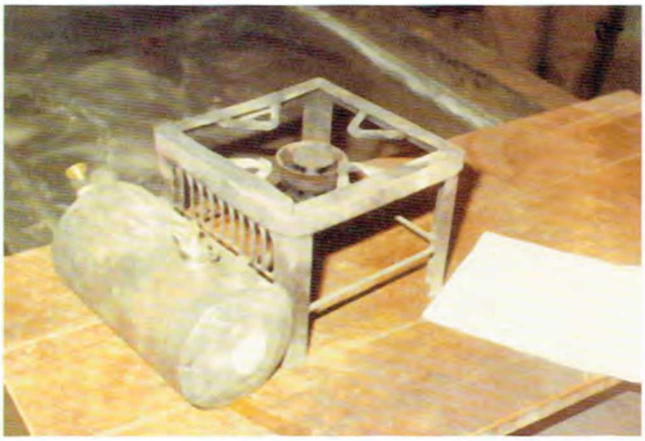

Fig. 35.3C: In the same case nonfunctional kerosene oil stove allegedly blamed for accidental burns

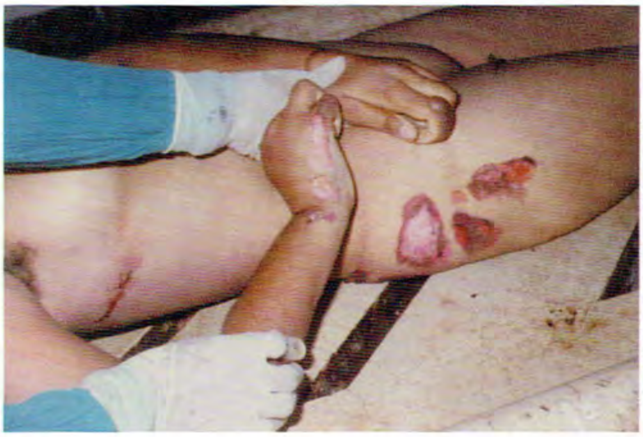

Fig. 35.4A: Case of dowry burns with strangulation – Deceased showing antemortem scalds on left index finger and dorsum of right hand and hot metal antemortem burns on right thigh indicating torture prior to strangulation

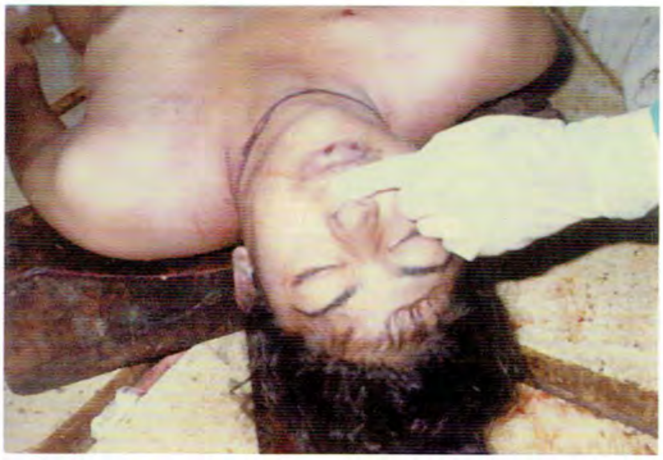

Fig. 35.4B: Same case with antemortem cigarette burns on lower lip and right side of chest also indicating torture

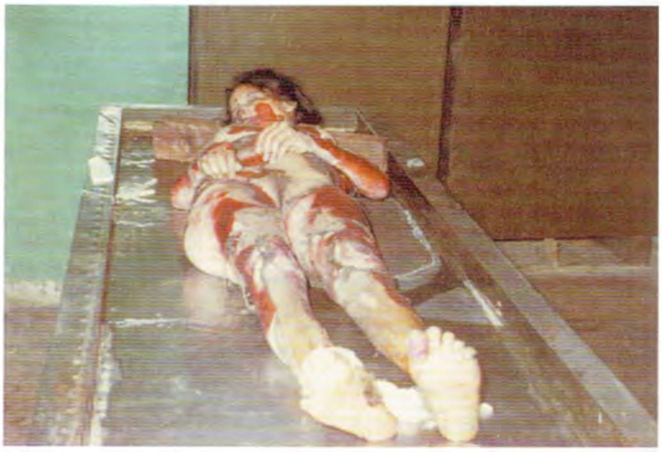

Fig. 35.5B: Same case with ink impression on right big toe suggestive of recording of statement of the deceased prior to death

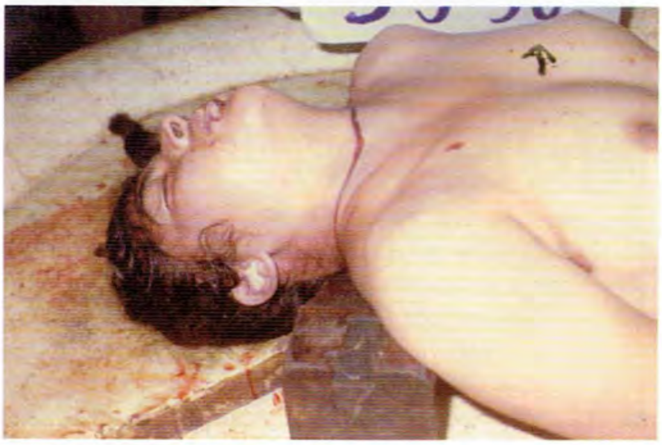

Fig. 35.4C: Same case with deceased showing sign of strangulation on neck

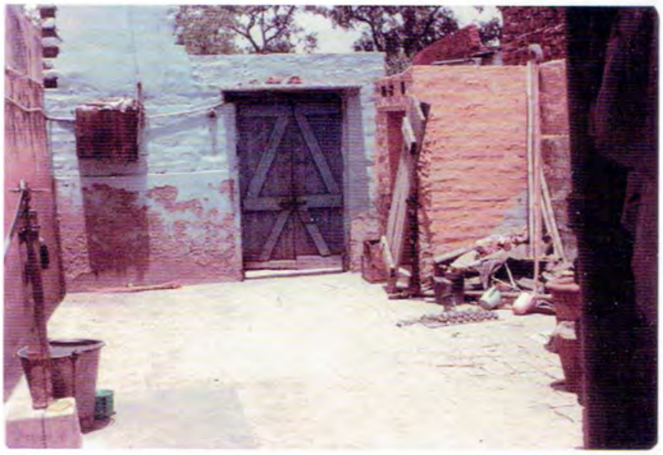

Fig. 35.5C: In the same case, visit of the scene of crime by the autopsy surgeon who conducted the postmortem — Showing door locked from inside

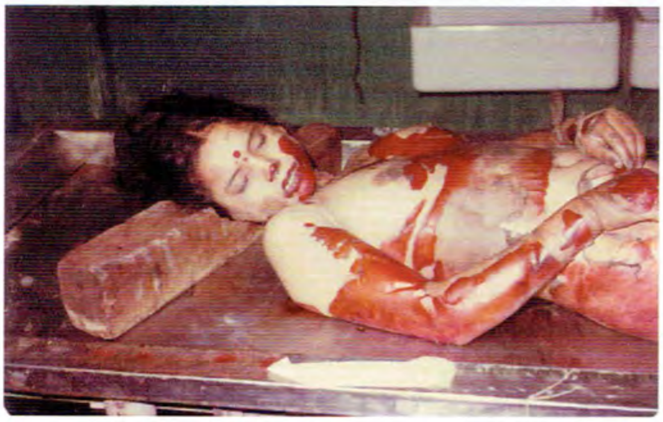

Fig. 35.5A: Typical antemortem homicidal burns with normal hair and scalp

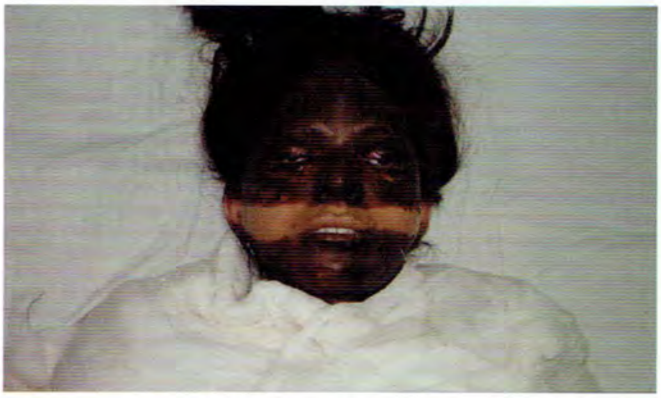

Fig. 35.6: Typical case of 100 % homicidal burns showing intact skin as a white band on the face with surrounding burned skin indicating that the patient was gagged before setting her on fire

on the head and also other parts of body and then body is set on fire producing characteristic features like burning/singeing of scalp hairs, eyebrows and eyelashes, burning or charring of scalp skin, ears, ear lobes or temple region.

Homicidal Burns (Figs 35.4A to 35.6)

Cases of homicidal burns are rare. Generally kerosene oil, petrol, diesel, alcohol, hot metals or corrosive substances have been used to inflict burn injuries. There is an increasing trend of homicidal burns in India with the sole intention of taking revenge or concealing the crime of murder and accordingly bodies are burnt to destroy these evidences. A detailed postmortem examination including evaluation of circumstances, even a visit to scene of crime will help in evaluating the nature of burn injuries and cause of death.

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Chapter

36

Legal Requirements in Managing Burn Patients

Atul Kumar Shah

DISCLAIMER

Foregoing presentation is a personal view of the author, based on his personal experience. Any future legal amendments may change these views.

INTRODUCTION

Burn accidents usually take place due to failure to prevent. This trauma has an element of suspicion of a crime added to it in many cases. A treating physician has the responsibility of informing such incidences to legal authority.

Police is constantly engaged in keeping law and order in control. Society needs to support police in maintaining peace and in reporting crime. Police actually has to classify any mishap as crime and subsequently investigate the same. For collecting information, police has to depend on various channels; one of them naturally is the body of citizens. Casualty doctors or treating physicians are duty bound to inform the police when they are managing trauma victims. Although this chapter deals with burn management and legal requirements in treating a burn victim, same procedures are equally applicable to other trauma, poisoning, drowning, suicidal and homicidal victims.

INDIAN LEGAL SCENE

Indian Penal Code (IPC) enlists various body injuries. Code of Criminal Procedures in section 39 asks every citizen to inform police of any incidence which has harmed any human being. For treating physician, informing police is, thus, an extension of his duty as a citizen. To the best of knowledge of the author, there is no requirement or compulsion specified in Medical Council of India Act or Bombay Police Act and the like.

SOCIAL CIVIC AND MORAL DUTY

Except as required under section 39 (section 40 for village head, police Patel or a villager) of the criminal Procedure Code, the need for informing the police in case of a suspicious burn injury is social as well as moral rather than legal. However, current sociolegal set up demands that police should be informed, whenever a trauma or burn case of nature as enlisted here, is received by a treating doctor.

SITUATIONS

All major burns and other major trauma victims when received have to be notified to the police. Unexplained severity, not matching with the history or circumstances, needs reporting. Patients received after several days of burns and without proper treatment, patients likely to succumb to the injury, patients received dead and mass casualties need to be notified to the police.

Usually when a patient succumbs to injuries, a certificate of death is required to be issued. In this certificate the cause of death has to be specified. When the treating doctor is not certain about the cause of death, demand of a medicolegal postmortem has to be made. Such a demand can only be made through a police officer. When the police officer is informed of such a request his team has to visit the site where death has occurred. He carries out a report of the condition and situation of the body, known as 'panchnama'. He also fills an inquest for a medicolegal postmortem. Later, the body is handed over to this police officer, who takes it to nearby health establishment equipped with postmortem facilities. If the inquest has been filled in odd hours, the body is shifted to a morgue for postmortem later or else the body may be retained, at the request of the police officer, in the same room until shifted for postmortem next day morning. The police enter the scene and fill inquest without wasting time; only if, they had been informed when the patient first came for treatment or got admitted.

Sometimes, the patients come from distant places and as per the police codes, the police officers of the area of incidence are required to carry out legal procedures. It is important to inform the nearby police immediately, so that at the time of the death of the patient, the nearby police do not waste time in procedural delays.

Similarly, in patients at extremes of age, extensive burns and evident homicide or suicide, it is better to inform police, as there is likelihood of death and subsequent postmortem. All road traffic and industrial accidents are also to be reported to police and are governed by the Motor Vehicle Act and the Factories Act. It is the duty of the treating doctor, in his capacity as a citizen or as a person with the information of the mishap, to inform police.

HOW SOON TO INFORM?

Supreme Court of India in a judgment has clarified that police activities can wait, the life of the victim is important and hence the treatment must be instituted immediately. Some judgments talk of 'reasonable time'. Therefore, one can conclude that as soon as the emergency care is over and the patient is stabilized, the process of informing the police may be started. Although it is not specified any where, we must make efforts not to make any delay in informing the police and this can be considered as within 4 to 6 hours or in case of very specific situations within 12 hours. Delays further than that may arouse suspicion in the mind of the investigating officers and their co-operation may get reduced.

WHOM TO INFORM?

This can be looked at from two different angles. In large cities there are several police stations with area demarcations, it may so happen that the burn incident may have occurred in the jurisdiction of one police station and the treating hospital may be in jurisdiction of another police station. If the police station in which the area of accident falls is willing to take the information on phone or if it is not far away and written information can be sent without extra efforts, the same may be done. Doctor can decide to inform the nearest police station as an alternative.

The local police station may not be willing to take such information if the area of incidence is different and many times the approach of the local police station is variable depending on the current officers and their superiors. In case of such difficulties, in many places one can utilize services of police control number 100. Here, telephonic information can be given and it is one of the legal ways of registering information. From Suburban areas also one can call the district Police Control Room and the connecting number is usually the same.

The burn victim comes from far off places, which may fall in other districts and even other states. Treating doctor or hospital may not invest time in trying to find out contacts of the exact police station of the mishap area for passing on the information. The nearest police station, under whose jurisdiction the treating hospital falls, must, however, be informed without fail. If, for some reasons, the nearest police station shows inability to accept the information, informing police control at 100 is a legal alternative. At many places, police has announced e-mail addresses for accepting such information.

MODE OF INFORMATION

Calling the police station on phone and giving brief particulars of the victim as well as of the incidence is the accepted way of intimation now at most of the places. Each police station has one 'on duty' PSO (Police Station Officer) that usually attends the phone and writes down the details. This information is then passed on to the 'on duty' Investigating Officer. The doctor is required to obtain the name of the PSO and ask him as to what time of intimation he has entered in his records. Later, the police station converts this information into their procedural information and enters this into a special register which they call 'station diary'. On shifting the information to station diary, a diary number or FIR (First Information Report) number along with the date and year is generated and it may be of help for subsequent communication with the police.

Sometimes, the police insists on a written information, in which case if it is feasible, a letter stating name, age, gender, and address of the victim, address where mishap occurred, time when mishap occurred, time when patient arrived to current health establishment and whether police has been informed by any other source(s) at any other health establishment(s) treating the patient previously, must be sent in duplicate so that proof of receipt can be obtained. As an alternative, such a letter must be prepared in three copies and two copies are handed over to a willing relative of the patient, taking his signature on the third copy stating that he has received a letter for informing the police. The relative then goes to the police station and gets signature of the PSO on the duplicate, which must then be received back in the hospital. This exercise is necessary as it keeps a proof with the hospital that a process of informing the police has been initiated and it saves the hospital staff from running around. The larger and designated government hospitals have a satellite police post inside the hospital and the information process is managed by specially appointed casualty doctors.

In case of paucity of time and manpower, unwilling relatives, unattended patients and when the nearby police station or the police station of the mishap area have not been reached by phone, call to police at control room number 100 would be sufficient. One must not forget to obtain the name of the person who attends your call at police control room and must register the time of such intimation in the case paper at appropriate place.

Police Control Room later identifies the responsible police station and transfers the information. Team from concerned police station visits the hospital and takes note of the mishap as well as the health status of the victim.

BURN INJURIES IN MARRIED FEMALE

When a married woman is brought with extensive burn injuries, the police tend to treat this mishap as a dowry problem unless proved otherwise with circumstanial evidence. If the married life of the victim at time of mishap is less than seven years, the police investigation becomes stringent and under a senior officer of the rank not less than a deputy commissioner. Death in such cases would be governed by section 304B of Indian Penal Code that specifies dowry deaths and can lead to imprisonment ranging from seven years to life.

CRIMINAL NEGLIGENCE

In a recent Supreme Court Judgment, guideline has been set up before applying section 304A against doctors. Rash or negligent act leading to death can attract imprisonment of up to two years and fine. However, the apex court has asked the investigating officers to first obtain sanction from senior government doctors to establish negligence in a particular case before arresting a doctor.

SUMMARY

Informing police is the duty of every citizen and so also of a doctor, when any mishap involving bodily injury is brought to their notice. With usage of electronic media, it has become less procedure oriented and less time consuming to inform police. Physicians treating burns must make sure that legal requirements have been properly met with.

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- Code of Ethics and Etiquette, Medical Council of India Act

Chapter 37

Burn Disfigurement and Physical Impairment Calculations

Atul Kumar Shah

INTRODUCTION

A burn victim is entitled to compensation for current and future medical expenses, lost wages, disfigurement, emotional sufferings, mental impairment and physical disability. The concept of physical impairment has to evolve amongst the burn specialists so that the award of disability can become more meaningful. It must be realized that disability is not purely a medical condition. Burn disfigurement brings unwanted stress and breaks down self-confidence and self-esteem. Burn injury victims also suffer excruciating pain as their wounds heal. Burn patients undergo painful skin grafting and other surgical procedures to repair the damage done by the injuries. Burn survivors may endure physical trauma, pain, body metabolism damage and changed temperature sensitivity. After the physical scars begin to heal, the emotional scars begin to surface. As a wild guess based on the experience of the fellow burn specialist colleagues, approximately one million burn injuries could be occurring in our country per year. Approximately half of these injuries are treated by medical professionals; about one tenth of them are hospitalized. Of those hospitalized many have major burns involving at least 25 % of their total body surface. Between 20,000 and 25,000 of patients with burns die and approximately half a million sustain substantial or permanent disabilities resulting from their burn injury. There must be very extensive and collective effort to bring out the burn incidence data in the country and use of census may also be taken to determine prevalence of disfigured individuals.

Burn accidents may be caused by someone else's carelessness or failure to act. Many a times, it is a combination of negligent act or a failure to act by one or more persons or entities that causes an accident to occur. Burn injuries can result from a variety of different activities and sources. Whether at home or on the job, one can be exposed to unknown dangers. The law requires that each person owes every other person a duty to act as a reasonably prudent person would act under the same or similar circumstances. When a person or entity does not act reasonably, then they have violated that duty and will be held responsible for any injury or damage that results. This duty is created from statutory laws or prior judgments of the cases that have evolved over time.

As a trauma or burn specialist we must have some understanding of the legalities of the award of disability by various competent authorities. In India, currently we have to depend mainly on the Workmen's Compensation Act of 1923 and the Motor Vehicle Insurance Act to understand the concept. Physical Impairment may translate into limitations of functional capacity of any individual and these limitations may be termed as 'disability to sustain' or 'disability to earn livelihood'. The definitions from WHO may make this point clear.

The chapter describes the difference between concepts of physical impairment and disability and guidelines to quantify permanent physical impairment.

Impairment and Disability

Impairment is permanent or transitory, psychological or anatomical loss and/or abnormality. Paralysis, amputation, diabetic retinopathy and even burn disfigurement or contracture are physical impairments.

Functional Limitation is partial or total inability to perform the activities necessary for motor, sensory or mental functions within the range and manner of which the human being is normally capable such as walking, lifting loads, seeing, speaking, reading, writing, counting, taking interest in and making contact with surroundings. Functional limitations come from physical impairment and may last for a short time, a long time, be permanent or reversible. Idea is that, whenever possible, one must be able to quantify these functional limitations. So these limitations are described as regressive or progressive.

Disability is difficulty that exists in performing one or more activities. These are the 'Activities' that in accordance with the age, gender and normative social role of the person are generally accepted as essential and basic components of daily living such as self-care, social relations and economic activity. Physical Impairment and functional limitations are the causative factors for this disability. Also depending in part on the duration of functional limitation, disability may be permanent, long-term or short term. We may, however, be dealing with Permanent Physical Impairment (PPI) and Permanent Disability most of the time.

Recently, WHO has proposed revision of the earlier definitions and has given new terminologies like 'activity limitation' and 'participation restriction'. Activity is the nature and extent of functioning at the level of the person and may be limited in nature, duration and quality. Participation is the nature and extent of a person's involvement in life situation and the restriction is in relation to impairment of activities, health conditions and contextual factors and participation may be limited in nature, duration and quality.

According to Webster's Dictionary, disability is defined as:

1. "State of being disabled: Absence of competent physical, intellectual, or moral power, fitness or the like; also an instance of such lack".
2. "Legal incapacity, incompetence, or disqualification".

There are physical, social, psychological and vocational effects to disability as well. A trauma specialist is, thus, not equipped and qualified to determine disability of a person, since disability is not purely a medical condition. Medical doctors must, therefore, restrict to evaluating and certifying physical impairment. In doing so the medical expert wishfully thinks that the competent authority shall subsequently consider the social, psychological and vocational aspects in calculating the disability of the individual in question. Unfortunately, currently the compensation authorities or the courts of law have limited understanding to demarcate between physical impairment and disability. Facial burns may not be considered a disability at all, but how can such a person ever become a marketing executive or a model or a TV news reader! On the other hand, a paraplegic is considered highly disabled, but he earns fortune by his vocal performances or lecturing on stress management and also qualifies for the concessions available to a disabled!

Under the Workmen's Compensation Act, disability may be divided into three periods.

Permanent disability shall apply to permanent damage or to loss of use of some part of the body after the stage of maximum improvement has been reached and the remaining physical impairment is stationary; this physical impairment shall then be termed as permanent physical impairment. There is a question of time frame here, and one must allow at least six months, if not more, before quantifying permanent physical impairment.

Temporary disability can be total or partial.

Temporary total disability shall be the period during which the individual is under treatment and totally physically impaired for the time being.

Temporary partial disability shall be the period when the physical impairment is partial to begin with or the person has recovered from the total physical impairment.

Ultimate legal compensation may be awarded, usually, for the permanent physical impairment of non-reversible nature leading to permanent disability. A medical person, thus, needs to evaluate permanent physical impairment and may even be called in the court of law to testify as an expert witness. Physical impairment certificates can be issued by all medical graduates who are registered under the schedule I of Medical Council of India Act 1956, and there is no need for the medical graduate to be a specialist, as is some times demanded by the lawyers. However, the court always considers the specialization and the expertise of the certifying doctor while relying on the assessment of physical impairment certified by the doctor.

In the United States, the American Medical Association's Guides to the evaluation of permanent impairment is the most commonly used source for assessing and rating an individual's permanent impairment. The fifth edition of the "Guides" (Chicago, Ill: American Medical Association: 2000) defines impairment as "the loss, loss of use, or derangement of any body part, organ system or organ function". Impairments are defined at an organ system level with objective measures such as lung or cardiac function.

Impairment ratings, based on medical consensus, were designed to reflect the ability to perform basic activities of daily living (e.g. personal hygiene, communication, standing, walking, sleeping and social and recreational activities). Typically, impairment ratings do not account for the effects of accommodations (exceptions include the use of glasses for assessing vision).

Impairment ratings in the "Guides" attempt to link objective measures of organ function with the functional tasks of performing activities of daily living, recognizing that there is often insufficient scientific evidence to support these ratings. Estimating work ability from an impairment rating can be even more problematic. Although these activities of daily livings are components of many workplace activities, work is a complex and individualized activity. Thus, the impairment ratings do not reflect an individual's ability to work, although they may be used to assess some components of work ability. Disability is an alteration of an individual's capacity to meet personal, social, or occupational demands because of an impairment or functional limitation. The "Guides" does not directly address disability but talks of work ability!

In India, apart from the Workmen's Compensation Act, the only other authentic documents available till date, to the best of knowledge of the present author, are 'The Manual for Doctors to evaluate Permanent Physical Impairment[1] and 'Evaluation of Impairments, Disabilities in case of Occupational and other Diseases and Accidents'.[2] The contents of the Manual for Doctors to evaluate Permanent Physical Impairment are based on the experts' group meeting on Disability evaluation that was held in New Delhi in September 1981 and also the National Seminar on Disability Evaluation and Dissemination held in New Delhi in December 1981. The Manual is endorsed by Director General Health Services (Govt of India), All India Institute of Medical Sciences (New Delhi) and World Health Organization and was published in March 1982.[1] The evaluation criteria are simplified and can be of immense help in issue of certificates of Permanent Physical Impairment. Guidelines are given here for evaluation of Permanent Physical Impairment in Burns of Head and Neck (Table 37.1) and Burns of Trunk and Genitalia (Table 37.2).

Guide lines for evaluation of physical impairment in burns of upper extremity:

1. The estimation of permanent impairment depends upon the measurement of functional impairment and is not expression of a personal opinion.
2. The estimation and measurement should be made when the clinical condition has reached the stage of maximum improvement from the medical treatment. Normally this time period is to be decided by the medical doctor who is evaluating the case for issuing the Permanent Physical Impairment Certificate as per standard format of the certificate.
3. The upper limb is divided into two component parts; the arm component and the hand component.
4. Measurement of loss of function of arm component consists of measuring the loss of motion, muscle strength and coordinated activities.
5. Measurement of loss of function of hand component consists of determining the prehension, sensation and strength. For estimation of prehension; opposition, lateral pinch, cylindrical grasp, spherical grasp and hook grasp have to be assessed as shown in hand component section of Assessment Performa for upper extremity.
6. The impairment of the entire extremity depends on the combination of the functional impairments of both components.

For extremities, one can take help of The Manual for Doctors to evaluate Permanent Physical Impairment or the recently published guidelines from Ministry of Social Justice. (http://socialjustice.nic.in/disabled)

Detailed format for extremity evaluation can be obtained from http://socialjustice.nic.in/disabled/part2.htm

In Workmen's Compensation Act, the reward is based on the wages as well as the involved parts and a brief summary of the currently awarded compensation is given here in Table 37.3.

Plight of a Burn Survivor

When any earning member of the family sustains major burn injury, the entire family receives the crunch. Unlike other trauma or disease, the management of burns is prolonged, painful and costly. The economic resources of the family get exhausted with the extent of treatment. Industrial burn accident victims are no exception. The burn survivor has to face challenges constantly even after the initial assault is over.

1. Physical and emotional trauma
2. An injury that leaves one in pain
3. Disfigurement
4. Organ damage
5. Metabolic and biochemical damage
6. Sensitivity to temperature change

Table 37 .1: Guide lines for evaluation of physical impairment in burns of head and neck. Ten point formula for evaluating post burn disfigurement of head and neck. Total 100 points and eight equitable components

Component	*Equitable Points*	*Split up Points*
1. Scalp and Vault (10)	10 (Includes Forehead)	
a. Scalp(Disfigurement alone)	2.5	
b. Scalp and Skull Bone Involvement	10	
2. Eye Brows (10)	5 each, 10 for both	
a. Part of One	2.5	
b. Total Loss of One	5	
3. Eye Lids (20)	6 for Upper, 4 for Lower	
a. Upper - Skin Disfigurement alone	Per Side	1.5
b. Upper - Deformity or Full Thickness Loss	Per Side	6
c. Lower - Skin Disfigurement alone	Per Side	1
d. Lowerer - Deformity or Full Thickness Loss	Per Side	4
4. Pinna (20)	10 per side	
a. Anterior or Posterior - Skin Disfigurement Alone	Per Side	2.5
b. Deformity due to full thickness involvement of skin and cartilage without obliteration of meatus	Per Side	7.5
c. Deformity due to full thickness involvement of skin and cartilage with obliteration of meatus		10
5. Nose (10)	10	
a. Skin Cover Disfigurement alone		2.5
b. Deformity due to full thickness involvement with both Nares Patent		7.5
c. Full thickness deformity with one nare obliterated (7.5 +1.25)		8.75
d. Full thickness deformity with both nares obliterated		10
6. Cheek and Lateral Area of Face(10)	5 Each	
a. Skin Cover Disfigurement alone	Per Side	1.25
b. Deformity or Full Thickness Loss	Per Side	5
7. Lips - Upper or Lower (10)	5 Each	
a. Lips - Upper or Lower skin cover disfigurement	Per Side	1.25
b. Lips - Upper or Lower skin cover disfigurement crossing midline		2.5
c. Deformity or full thickness loss of one lip alone		5
d. Deformity due to involvement of both lips leading to contracture of oral opening		10
8. Neck (10)	10	
a. Skin Cover Disfigurement alone	Per Side	2.5
b. Deformity due to involvement of skin, muscles or deeper tissues		10

Table 37.2: Guidelines for evaluation of physical impairment in burns of trunk and genitalia

1. Front of Trunk and Abdome excluding Breasts	Female	10	Male	5
2. Breast each Side total or as per detail below	Female	20	Male	5
a. Only Skin Cover disfigurement each side	Female	5	Male	1.25
b. Involvement of Skin, Areola and Nipple and/or Deformity resulting in loss of function - per side	Female	15	Male	5
c. Involvement of Skin, Areola, Nipple and parenchyma and / or Deformity resulting in loss of function- per side	Female	20	Male	5
3. Total Back	Female	5	Male	10
4. Groins - each side	Female	5	Male	5
5. Buttocks - each side	Female	2.5	Male	2.5
6. Genitalia total	Female	30	Male	60
a. Skin loss resulting in mild deformity	Female	7.5	Male	15
b. Severe contracture of orifices or sloughing of urethra or severe deformity of penis	Female	30	Male	60

Table 37.3: Indicative Compensation payable currently under the Workmen's Compensation Amendment Act (46 of 2000)

	Instance	*Minimum Compensation*
a.	Where Death result from Injury	Rs. 80,000
b.	For Permanent Total Disability	Rs. 90,000
c.	For Permanent Partal Diablement	As per % of loss of earning capacity. It varies from 1 % to 90 %
d.	Temporary Disablement	An amount equal to 50 % of monthly wages subject to maximum Rs. 1000. For up to 3 days disablement, no compensation is payable. But if it is more than 3 days but less than 28 days, then 50 % of the balance days wages are payable. If it is more than 28 days, then the 3 days are not deducted.

After the 2000 amendment, the maximum liability under the act for workman is limited to a monthly wage of Rs. 4000. In such cases the compensation based on actual last monthly wage is as follows.

a.	Where Death result from Injury	An amount equal to 50 % of monthly wages of the deceased workman multiplied by the relevant age factor or Rs. 80,000 - whichever is more.
b.	For Permanent Total Disability	An amount equal to 60 % of monthly wages of the deceased workman multiplied by the relevant age factor or Rs. 90,000 - whichever is more.

Legal Rights of Burn Survivors to Compensation

Highly dependant upon the prevailing legal and social support, the following compensation can be claimed by the burn victim depending on the cause of the burn injury. Compensatory damages "compensate" the injured person for various kinds of losses or damages and include - current medical expenses, lost wages, anticipated future medical expenses, anticipated future loss of wages, mental or emotional pain and suffering (past and anticipated in the future), disfigurement and any physical or mental impairment or disability.

I. Payment for treatment—Past and future
   1. Hospitalization
   2. Surgical procedures
   3. Ongoing medical care
   4. Counseling
   5. Scar revisions—Cosmetic surgery
   6. Physical therapy
   7. Occupational therapy

II. Compensation for loss of income—past and future

III. Vocational Rehabilitation (job retraining)

IV. Compensation for pain and suffering—Burn survivors may be entitled to compensation for the pain and suffering that they have endured and may continue to endure as a result of their injury

V. Loss of Consortium—This is a novel concept for Indian Judiciary and may soon find its place in the case laws. The Spouses of a burn survivor may be entitled to compensation when an injury is so severe that it interferes with the injured party's spousal relations. The affected family member may suffer a very real detriment. Many courts recognize the right of the injured party's spouse to recover in an appropriate case for a loss of support, services, love, companionship, society, affections, sexual relations and solace in the form of a loss of consortium action. Loss of consortium is a claim separate from the injury victim's claim. It is unique to the injured party's spouse and is compensable by a separate damages recovery.

Indian Legal Enforcements

Burn survivors with untreatable contractures, keloids, grafted area, disfigurement and the like can be qualified to receive state help. Burn and trauma experts need to put up united front in making these benefits available to burn survivors. The enactment of the "Persons with Disabilities (Equal Opportunity, Protection of Rights and Full Participation) Act, 1995 (referred to as Persons with Disabilities Act), is a signal achievement of the Indian disability movement. Preamble to this Act clearly delineates its objectives of promoting and ensuring equality and full participation of persons with disability. The Act aims to protect and promote economic and social rights of people with disability. In Section 2(t), a person with disability means ' a person suffering from not less than forty percent of any disability as certified by a medical authority'. The disabilities that have been listed in section 2 include blindness, low vision, hearing impairment, loco- motor disability or cerebral palsy, mental retardation, mental illness and persons cured of leprosy. In addition, autism and multiple disabilities have been covered under the "National Trust for Welfare of Persons with Autism, Cerebral Palsy, Mental Retardation and Multiple Disabilities Act, 1999".

Prevention of Disability

The law in its present form only exemplifies a narrow understanding of the right to health with emphasis on prevention, cure, improvement or elimination of disability, which in any case are conditions that generally exist before the onset of disability and therefore, have no logical connections to the rights of those who are disabled. For example, Section 25 of the Persons with Disabilities Act, 1995, states as follows: "Within the limits of their economic capacity and development, the appropriate Governments and the local authorities with a view to preventing the occurrence of Disabilities, shall, (a) undertake or cause to be undertaken surveys, investigations and research concerning the cause of occurrence of Disabilities; (b) promote various methods of preventing disabilities; (c) screen all the children at least once in a year for the purpose of identifying 'at-risk' cases; (d) provide facilities for training to the staff at the Primary Health Centers; (e) sponsor or cause to be sponsored awareness campaigns and disseminate or cause to be disseminated information for general hygiene, health and sanitation; (f) take measure for prenatal, perinatal and postnatal care of mother and child; (g) educate the public through the pre-school primary health centers, village level workers and "anganwadi" workers; and (h) create awareness amongst the masses through television, radio and other mass media on the causes of disabilities and the preventive measures to be adopted.

As certain groups among the disabled are more vulnerable than others, a special enactment for the protection of such persons, their property and well-being was felt necessary. The enactment of the National Trust for Welfare of Persons with Autism, Cerebral Palsy, Mental Retardation and Multiple Disabilities Act 1999 (referred to as the National Trust Act) aims to fulfill a common demand of families seeking reliable arrangement for their severely disabled wards. The specific objectives of the Act are:

1. To enable and empower persons with disabilities to live as independently and as fully as possible within and as close to the community to which they belong;
2. To promote measures for the care and protection of persons with disability in the event of death of their parent or guardian; and
3. To extend support to registered organizations to provide need based services during the period of crisis in the family of disabled covered under this act.

The National Trust Act mandates the creation of a local level committee (LLC) comprising District Magistrate along with one representative from a registered organization and one person with a disability.

The Rehabilitation Council of India Act, 1992

The Rehabilitation Council of India was set up by the Government of India in 1986 initially as a society to regulate and standardize training policies and programmers in the field of rehabilitation of persons with disabilities. The urgent need for minimum standards was felt as the majority of persons engaged in education, vocational training and counseling of persons with disabilities were not professionally qualified. Poor academic and training standards adversely affect the chances of disabled succeeding in the world of work. Therefore, an Act of parliament in 1992 enhanced the status of the council to a statutory body with following aims:

1. To standardize training courses for professionals dealing with people with disabilities;
2. To prescribe minimum standards of education and training for various categories of professionals dealing with people with disabilities;
3. To regulate these standards in all training institutions uniformly throughout the country;
4. To promote research in rehabilitation and special education; and
5. To maintain Central Rehabilitation Register for registration of professionals.

The Rehabilitation Council of India (RCI) regulates training standards for sixteen categories of rehabilitation workers. The Council is pro-actively promoting training and research initiatives utilizing experience of specialized as well as mainstream academic institutions.

Ministry of Social Justice and Empowerment had amended section 47 of the Persons With Disability (PWD) Act regarding non-invalidation of the government servant who has been permanently incapacitated on account of mental or physical disability vide order 6/17/2002-SCS/1925 and states that no establishment shall dispense with or reduce in rank an employee who acquires a disability during his service and the employee who has acquired disability, if not suitable for the post he was holding, could be shifted to some other post with the same pay scale and service benefits. In case it is not possible to adjust him against any post, he may be kept on supernumerary post until a suitable post is available or he attains the age of superannuation, whichever is earlier. No promotion shall be denied to a person merely on the ground of his disability.

According to the persons with Disabilities Act rules, Ministry of Social Justice and Empowerment issued a gazette notification on June, 1, 2001. Here guidelines for evaluation of following disability and process of certification are outlined (i) visual disability, (ii) loco-motor / orthopedic disability (iii) speech and hearing disability, (iv) mental retardation and (v) multiple disabilities. Medical Board duly constituted by the Central and State Governments can only issue disability certificate. The State Government can constitute a medical board consisting of at least three members out of which at least one shall be a specialist in the particular field, as the case may be. The Director General of Health Services, Ministry of Health and Family Welfare, Nirman Bhawan, New Delhi will be the final authority, should there arise any controversy/doubt regarding the interpretation of the definitions/classifications/evaluation tests and the like.

SUMMARY

Quantifying the Permanent Physical Impairment and issuing authenticated certificate for the same has been facing cross questions from the advocates. There is limited understanding about difference between physical impairment and disability amongst medical and non-medical fraternity. Medical personnel are empowered by the MCI act to issue certificate of Permanent Physical Impairment based on the guidelines prevailing in the country at present. It then becomes the duty of the Law or compensation authorities to calculate loss of earning capacity of the person. Guidelines for calculating physical impairment in burns of head and neck, face, trunk and genitalia are based on the area and depth of involvement and calculation can be so based easily in arriving at the percentage of permanent physical impairment of a particular anatomical component of the body. There are several efforts at both the national and international level to help and support disabled persons and burn survivors would qualify for several of these services.

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7

Section

Organization of Burn Care

Chapter

38

Organization of a Burn Unit

Madhuri Gore

INTRODUCTION

Optimal functioning of a burn unit demands commitment from the burn team and the hospital administration. A specialized burn unit can contribute significantly to the field of burn care as it offers following advantages—

1. Facilitates accrual of experience.
2. Permits implementation and evaluation of protocols.
3. Facilitates collection and analysis of data.
4. Provides stimulus for innovation and research.
5. Provides facility for training.
6. Opportunity to improve the quality of burn care leading to reduction in mortality and morbidity.

The main purpose of a burn unit is to minimise the incidence of infection in burnt patients and to provide comprehensive burn care. The design of a burn unit should be conducive for this purpose. A typical burn unit should have provision for 11-18 patients.[1] The actual number of beds depends on the available data about patient load.

MEMBERS OF BURN TEAM

The core of the burn team is formed by:

1. Unit Director - Burn surgeon – General or plastic surgeon with one year fellowship in burn care and at least 2 years experience.[2] Junior consultant surgeon and training surgeons complete the surgical contingent.
2. Nurse Manager – with at least two years experience in acute burn care and 6 months of managerial experience.[2] Nursing staff and trainee nurses complete the nursing team.
3. Microbiologist
4. Nutritionist
5. Physical and occupational therapists, respiratory therapists.
6. Clinical psychologist
7. Social worker.
8. Labor staff.

The specialists from following departments should be available for consultation:

General Surgery, Orthopedics, Cardiothoracic Surgery, Otorhinolaryngology, Neurosurgery, Urology, Ophthalmology, Obstetrics/Gynecology, Internal Medicine, Pulmonary Medicine, Anesthesiology, Radiology, Pediatrics, Nephrology, Psychiatry, Neurology, Cardiology, Pathology, Gastroenterology, Microbiology, Hematology.

CASUALTY

The patient receiving area may be a part of the burn unit or of the Accident and Emergency department of the hospital. It should be easily accessible and should have an identified attending medical professional available 24 hours.

Facility size: A 12 feet × 12 feet area is comfortable for accommodating patient trolley, personnel and equipment.

Equipment: Intensive care patient trolley, crash cart with life saving equipments and drugs are essential components of the receiving area.

Weighing facility should be available.

Central suction and oxygen supply is desirable. A suction machine and oxygen cylinder with regulator and humidifier may be an acceptable alternative.

The instrument sets for the following procedures should be available:

1. Intravenous access including venesections and blood collection for investigations.
2. Tracheostomy.
3. Escharotomy/Fasciotomy
4. Insertion of nasogastric tube
5. Perurethral catheterization.

A dressing trolley with dressing materials in adequate quantity should be available if the receiving area is a part of burn unit. If it is in Accident and Emergency Department, the patient can be wrapped in a sterile drape and transported to the burn unit for further wound management.

The following documents should be displayed or be readily available in the receiving area:

1. Burn unit admission criteria
2. Protocol for initial evaluation and management.
3. Chart for evaluation of extent of burn.
4. I/V access protocol
5. Contact telephone numbers.
6. Burn mass casualty action plan.

DESIGN OF BURN UNIT

By definition, burn unit is a part of a multispecialty hospital and should occupy either a complete floor or a part of it. Control of infection should be the basic consideration while deciding the distribution of space in a burn unit. In order to minimize the transportation of micro-organisms, the necessity for movement of health care workers, patients and waste material within the unit should be reduced. This can be achieved by:

- Allocating zones within the unit for each category of patient condition (acute, infected, high dependency, non-acute / convalescent) and equipping each zone with all the patient-care requirements for daily routine (therapy, hygiene, staff areas, etc.). Depending on the available space, convalescent care area may be located outside but in physical proximity of the acute and high dependency care area of burn unit
- Keeping immunosuppressed, infected and acute patients isolated in single rooms;
- Employing appropriate measures for environmental control
- Organizing unidirectional traffic routes and corridors so that during routine procedures there are no points of contact between the various zones.

A concentric design with central nucleus of patient support and general service area; intermediate band of patient care rooms; external visitors' corridor has been recommended.[1]

The service areas should include:

- A nurses' station
- A kitchenette / pantry
- An on-call restroom
- Staff changing room
- Stores
- An examination room for out-patients
- An operating suite, including a "clean" operating theater and a dressing room
- A conference/meeting room and a communication room are desirable if the space permits.

The patient care rooms can be of different designs depending on the type of patient. The number of beds in each category will depend on the average number of admissions (The numbers indicated here are considered appropriate for about 400 admissions per year).

a. The patient at high immunological risk (acute and infected): 4 + 4 = 8 beds
b. The patient without particular immunological problems and in non-acute but high dependency state, i.e. after complete resuscitation, between wound excision and closure procedures: 8 beds
c. The patient in convalescent phase with healing of more than 50 % of the burnt area till the time of discharge: 8 beds

Acute Care Area

For acute or septic patients a single room is considered essential. Ideally, to limit still further the patient's transfer and the consequent dispersal of organisms, each room should have an adjoining:

- WC with sluice disposal (separate for each room)
- Hydrotherapy room (one between two single rooms, accessed through an air-lock from each side)
- Filter/ante-room (separate for each room, positioned between bedroom and access corridor and acting as air-lock to the tub room) would be ideal.

The minimum desirable dimensions of the acute care room are 10 ft × 10 ft × 10 ft The room should have central oxygen supply and central suction facility. The equipments needed for providing acute intensive care are as follows:

1. Intensive care bed – 1
2. A set of resuscitation equipment including laryngoscope, endotracheal tubes, Ambu bag, connections, suction cannulae, emergency drugs.
3. Multipara monitor / capnometer / cardioscope with pulse oxymeter (depending on availability of funds) - 1
4. Syringe infusion pump – preferably 2, at least 1.

5. Nebuliser – 1
6. Dressing trolley with dressing drum.
7. Stand-by suction machine.
8. Patient care articles such as bedpan, kidney tray, etc.
9. Hand disinfection facility should be provided in the ante-room or in the patient room in the absence of ante-room.

The acute care area should have

1. Sophisticated ventilators—Ideally 8 (one for each ICU bed), but at least 2, along with the required accessories.
2. Intensive care trolleys—2.
3. Crash cart—1.
4. ECG machine—1.
5. Portable X-ray machine—1.
6. Multipara monitor in each room connected to central monitoring station at nurses' station and closed circuit television facility in each room for observing the patient from nurses' station would be an asset that can be procured at a significant cost.
7. Vacuum cleaner – Heavy duty—1.

Surgical residents: 2 surgical residents should be assigned to this area.

Nurse: Patient ratio: 1: 1 very desirable for all shifts.

A dressing assistant is necessary for morning shift. Helper staff of minimum 4 ward attendants and 4 cleaners is necessary for every shift.

High Dependency Area

A room with two beds is desirable, with WC and shower for each room. At least 5 feet should be available on either side of the patient bed for ease of procedures and infection control. The equipment that should be available for each bed is:

1. Intensive care bed.
2. Central suction and oxygen facility.
3. A set of patient care articles.
4. Dressing trolley with dressing drum.
5. Splints, extremity supports.

Each room should have:

1. Pulse oxymeter – 1
2. Nebuliser – 1
3. Wheel chair – 1
4. Syringe infusion pump – 1
5. Volumetric infusion pump for constant rate nasojejunal tube feeds.
6. Simple physical therapy equipments.
7. Hand disinfection facility.

The high dependency area should have

1. Sophisticated ventilators – 2;
2. Intensive care trolleys – 2;
3. Crash cart – 1;
4. Physical therapy equipments;
5. Vacuum cleaner – Heavy duty – 1;
6. Washing machine – 1;

Surgical Residents: At least 1 surgical resident should be assigned to this area.

Nurse: Patient ratio: 1:2 for morning shift and 1:3 for other two shifts with patient allocation. A dressing assistant is necessary for morning shift. Helper staff of minimum 3 ward attendants and 2 cleaners is necessary for every shift.

The Convalescent Care Area

The convalescent care area should be in the proximity of ICU and HDU. It should have at least 8 beds. This can be a big hall with adequate space between the beds with facility to isolate the patient with curtains. Each bed should be provided with central suction and oxygen facility. Windows and exhaust fans can be used for providing good natural ventilation. At least 4 showers and WCs should be provided in the toilet block of the ward. Indian WC is unsuitable for burnt patients as squatting can be extremely difficult. Every bed should have a set of patient care articles such as chair, bucket, mug, and bedpan. Separate dressing drum for each bed is highly desirable and separate dressing trolley should be definitely considered. Use of patient cabinets should be avoided as these act as breeding areas for pests and insects. Instead a shelf can be provided for each bed for keeping personal articles of the patient. Hand disinfection facility should be provided for each bed.

This area should have service areas such as nurses' station, changing room, pantry, store, dressing room, physiotherapy and recreation area. The following equipments should be available in this area:

1. Patient trolleys – 2
2. Wheel chairs – 2
3. Crash cart – 1.
4. Nebuliser – 1.
5. Suction apparatus – 1.
6. Physical therapy equipment.
7. Vacuum cleaner – 1.
8. Music system
9. Television
10. Pulse Oxymeter – 1 – desirable
11. Syringe infusion pump – 1 desirable

Surgical Residents: At least 1 surgical resident should be assigned to this area.

Nurse: Patient Ratio: During morning shift when maximum patient care activities are carried out, at least 2 qualified nurses should be available along with one dressing assistant. In other two shifts at least 1 qualified nurse should be posted.

A dressing assistant is necessary for morning shift. Helper staff of minimum 2 ward attendants and 2 cleaners is necessary for every shift.

ENVIRONMENT CONTROL

The purpose of environmental control measures is:

1. To reduce the load of bacteria carrying particles
2. To impede the passage of air from areas with lower grades of asepsis (e.g. service areas) to those where the level of sterility is required to be high (e.g. patient room, dressing room) by maintaining pressure gradient.
3. To provide comfortable environmental temperature so as to help control the hypermetabolic state of the burn patients by reducing shivering.
4. To provide optimal humidity so as to control evaporative losses and to aid wound healing process.
5. To dilute the smells while keeping noise to minimum.

Air changes: Appropriately designed air treatment plant would serve this purpose by maintaining different levels of asepsis in the air according to the function of the space. The reduction of bacterial content in the air can be brought about by either filtration or sterilization. High efficiency particulate air (HEPA) filters have the smallest pore size, but its use entails high recurring expenditure. Filters with 3-5 micron size pores are considered adequate for ICU. The sterilization is effected using ultraviolet lamps positioned either directly in the room or in the air conditioning ducts as a complement to the filters.[1]

Three systems of high-efficiency cleansing are:

1. laminar flow
2. directed vertical flow
3. Joubert system.

Laminar flow systems exist with either horizontal or vertical orientation. In both cases the air-flow acts on the entire air volume and is characterized by its very high air-change rate (300-540 volumes/hour).

Directed vertical flow concentrates its action on the area in which the medical team is operating and in proximity of the patient. The system's capacity (20-25 changes/hour, which can drop to 15 when rooms are not in use), although notably lower than that of the laminar flow, is seen to reduce the content of Colony Forming Units (CFU) per cubic metre to a suitable level for delicate operations.

The Joubert system is a system based on directed vertical flow for operating rooms, introducing certain modifications: the air change rate is of around 50 volumes/hour; of these, 6 vol/H are fresh external air, while the other 44 are recycled from the room and filtered.[1] In the author's experience directed vertical flow system with 20-24 fresh air changes/hour reduces the CFU/cubic meter to less than 10 which is considered adequate. Regular maintenance of the system needs to be carried out by cleaning/changing of filters, cleaning of ducts and microbiological surveillance.

Temperature: Environmental temperature equal to normal body temperature i.e. 35°C is optimal for controlling the metabolic rate and to prevent shivering of burnt patients. This is most often perceived uncomfortable by the health care professionals as it causes sweating, fluid loss and easy fatigue. In the author's experience temperature of about 30°C is an acceptable compromise for both patient and staff if maintaining different temperatures in the patient rooms and service areas is not possible due to resource crunch.

Humidity: Humidity of 55-60 % is desirable.

Use of air curtains at identified entrances helps to maintain pressure gradient and thus aids control of the microclimatic conditions.

CONSTRUCTIONAL CONSIDERATIONS

The material used for floor and walls of the burn unit should be chosen with a thought about infection control, ease of cleaning and durability. The cost of the material could be a major deciding factor in presence of resource crunch.

The floor: Floor needs to withstand heavy load and traffic. It should be dust resistant, non slippery and easy to clean. Epoxy coating satisfies these criteria, but is expensive. Amongst the natural stones, marble is unsuitable as its high porosity supports trapping of moisture and growth of organisms. Granite is slippery and expensive. Polished Kota or polished Shahabad is strong, durable, affordable, easy to clean and with least porosity. Use of large size slabs would reduce the number of joints.

The walls: The walls should be covered from floor to ceiling with suitable material which should be dust resistant, non porous, easy to clean. Large, marbo-granite tiles are considered suitable as the porosity is less than ceramic tiles and the surface is dust resistant.

The ceiling: If false ceiling is planned, it should be of aluminium panels for ease of cleaning. If the ceiling needs painting, plastic emulsion or oil paint is recommended as both are moisture and dust resistant and easy to clean.

Cleaning: Use of vacuum cleaner is strongly recommended for cleaning of floor, walls and ceilings of all the areas in the burn unit. This sucks and removes the dust while sweeping displaces it.

The windows: If open ventilation is being used in the unit, the windows must have nets to prevent entrance of flies, mosquitoes and other insects. A strict protocol for cleaning of nets needs to be prepared and implemented.

OPERATION THEATER

The operation theater can be a part of the burn unit or it may be an identified OT in the surgical OT block which would save space and personnel.

1. Size – 20 feet × 20 feet
2. Operation table with facility to suspend extremities with the help of detachable rods or rails suspended from ceiling.
3. All usual OT equipment such as anesthesia machine, central gas supply and suction facility, cautery machine.
4. Thermal blanket, patient warmer, on-line fluid warmer, multipara monitor.
5. Air changes – 20-25 fresh air changes/hour, Temperature – 35° C, Humidity – 55-60 %.
6. Surgical team should have 2 surgeons, 2 assistant surgeons and 2 nurse assistants.
7. Special instruments – Skin grafting knife of choice. Dermatome is an asset, adequate number of blades, Skin graft mesher, dermacarriers.

DRESSING ROOM

During a dressing procedure, the bacterial density in the dressing room increases significantly. 8-10 air changes are needed to reduce the density to acceptable level. This would need a time gap of 30 minutes between two dressing procedures. In a busy burn unit it is impractical to follow this discipline. The author is convinced that doing dressings in the patient's own room is a convenient alternative. Selected dressings may be done in the dressing room with adequate time interval between the procedures. The dressing room should be of 12 × 12 feet for ease of manipulation of patient trolley. The environment control specifications would be same as for OT. It should have adequate supply of sterile dressing equipments and agents. A weighing machine can be conveniently placed in the dressing room for ease of implementing daily weight protocol.

HYDROTHERAPY FACILITY

A large number of burn units outside India use Hubbard tank for hydrotherapy. In author's opinion this is unsuitable for burn units in India for several reasons

1. The time taken for each patient is about an hour. Considering the number of patients in India, this would mean having multiple tanks which entails significant expenditure.
2. In the absence of appropriate cleaning of the tank, the risk of endogenous as well as cross infection is certainly increased. With constant paucity of trained and committed personnel, this is a definite possibility.

The experience in India as well as abroad suggests that shower bath (with hand held – telephone shower) using appropriately modified patient trolley is an effective alternative.[3] In contrast to the Hubbard tank, the trauma table was observed to have the following associated advantages: (1) quicker treatment sessions; (2) less pain to the patient; (3) elimination of patient exposure to contaminated water; (4) more comfort for the therapist; and (5) greater ease in patient handling, for example, rolling the patient onto a stretcher or bringing the patient into a sitting position. The use of the sterilized disposable plastic sheet was observed to be the most effective procedure to decrease the risk of contamination between patients during the shower.[4] The hydrotherapy facility should have adequate space and wide enough doors for manipulation of the trolley. Hot and cold water mixer with telephone shower attachment and appropriate drainage provision is necessary, inexpensive and easy to use and maintain. In ICU and HDU, trolley lined by sterile plastic drape is appropriate while in convalescent care area the patients may walk or be taken on a wheel chair for hydrotherapy.

It is important to note that hydrotherapy may not be safe for patients with hypovolemia, sepsis and hypothermia. For such patients it is advisable to perform sponging of non-burnt body parts and wound cleaning and dressing on the patient bed.

Provision must be made for appropriate collection and disposal of waste generated in the hydrotherapy area without contaminating the patient care areas. Maintaining this unidirectional movement is crucial for infection control and needs to be incorporated in the unit design.

PHYSIOTHERAPY UNIT

Physical therapist should visit the unit twice a day for passive and assisted and supervised exercises for every patient. For patients in ICU, splints and extremity supports should be available for optimal positioning. The extremity supports can be prepared from thermoplastic material or aluminium sheet (cheaper). Extremity supports designed at the author's unit cost about Rs. 200 /- for foot support and Rs. 400/- for upper extremity support. These have been observed to be effective and are easy to disinfect. Readily available aluminium splints for wrist and hand, elbow, ankle and knee help in providing splinting and are easy to disinfect.

The physiotherapy equipments that should be available in HDU and convalescent care area are

1. Cycle pedals fixed on a stand
2. Pulleys for upper extremity exercises.
3. CPM (Chronic Passive Motion) machines for different joint movements should be available, whenever needed.
4. Parallel bars can be provided in the unit if the space permits. The walker should be available.
5. Simple games such as peg in the hole, building blocks provide valuable exercise for fingers and wrists.
6. Toy cars and cycles provide excellent therapy to pediatric patients while they play.
7. In author's observation, group exercise therapy improves patient participation significantly. This can be effectively conducted in convalescent care area and for select patients in HDU.

It is necessary to ensure regular cleaning and disinfection of all the equipments. Besides the physical therapist, the nursing staff and surgical team should encourage the patients to perform activities of daily routine along with the exercises.

The physical therapists also have to provide respiratory therapy not only to the patients with inhalation injury but to all the patients in the unit. The necessary equipment (nebulizer, incentive spirometer) should be available in the unit and disinfection of the mouth pieces between patients should be ensured.

MONITORS

The details of desirable equipments in different areas of a burn unit have been provided earlier. The number and type of monitors would vary depending on the available resources. It is important to recognize the need to be vigilant and to use available monitors judiciously. The brain behind the eyes that look at the monitor screen is the most important equipment.

The possibility of outsourcing monitors and ventilators should be explored and a plan to do so should be ready and known to all involved.

Upkeep and maintenance of monitors, ventilators and infusion pumps needs to be done regularly to ensure optimal functional status.

UTILIZATION AND COST CONTAINMENT

Establishment of a burn unit is an expensive proposition. It should not be undertaken if optimum and appropriate utilization is not ensured. In our country, if a burn unit has to be closed for lack of patients, it would call for a celebration. But resource crunch is a major issue that plagues most of the burn units. To combat this problem we must find our own solutions based on sound scientific principles. The burn team should discuss various shortfalls openly and the team leader must encourage suggestions from each and every member. Imaginative innovations and their critical evaluation would certainly lead to development of methods that would take us ahead on the path of progress. The measures suggested here for environment control are expensive and available funds may not be sufficient. It should be realized that these are not solutions in isolation to the problem of hospital infections. These infections are the result of interacting causes, none of which should be undervalued or ignored and should be countered using interacting measures. For these reasons surgical and nutritional treatment and, above all, the conscientious respect of hygiene discipline on the part of all members of staff are of fundamental importance and cannot be substituted by any of the expensive gadgets. Large, well ventilated, multi-bed burn units can also generate impressive results with strict attention to cleaning protocols and barrier nursing. Excellent nutritional support can be provided using locally available inexpensive items of food instead of commercially available tube feeds or nutritional supplements. Paucity of funds should be considered as an opportunity to innovate. Burn care requires a high staff-to-patient ratio and a hospital budget to support this is mandatory. The author strongly feels that the resource crunch should not compromise the number of health care professionals in the burn unit –especially the nursing staff. Such a step is extremely detrimental to the quality of care and may result in loss of several valuable lives. This causes irreparable damage to the morale of existing staff, leading to further deterioration of services.

ACTIVITIES OF BURN UNIT

The burn unit should not be satisfied just with provision of burn care. The responsibility of the burn team and the unit extends much beyond to encompass the following.[2]

Performance Improvement Program

The burn unit should have a performance improvement program that is multidisciplinary.The burn unit multidisciplinary committee, which oversees the performance improvement program, should meet at least quarterly. Sufficient documentation should be maintained to verify problems, identify opportunities for improvement, take corrective actions and resolve problems.

Morbidity and mortality conferences should be held at least monthly with physicians other than the immediate burn care team to ensure objective review of the presentations. Attendees at this conference must include specialist staff members other than those practicing in the burn unit. All significant burn complications and all deaths must be discussed. There must be open, candid discussion with high points documented and assessment of the death or complication classified as "caused by disease," "potentially preventable," "possibly preventable" (or the equivalent thereof). Actions recommended must also be documented, and there must be documentation of loop closure. Records of this conference must be kept.

Patient care conferences shall be held at least weekly to review and evaluate the status of each patient admitted to the burn unit. Each clinical discipline should be represented in order to appropriately contribute to the treatment plan for each patient. Patient care conferences shall be documented in the progress notes of each patient and/or in minutes of the conference.

Audit

The burn service should conduct audits at least annually that should include, but should not be limited to, the severity of burn, mortality, incidence of complications and length of hospitalization.

Educational Programs

The burn unit should have an educational program for the medical and nursing staff.

If residents rotate on the burn service, the burn service director or his or her designee should be responsible for an orientation program for new residents.

Infection Control Program

The burn unit should have effective means of isolation that are consistent with principles of universal precautions and barrier techniques to decrease the risk of cross-infection and cross-contamination.

The burn unit should provide ongoing review and analysis of nosocomial infection data and risk factors that relate to infection prevention and control for burn patients.

Continuity of Care Program

The burn unit must provide the following services:

1. Recreational therapy for children cared for in the unit
2. Education in rehabilitation
3. Support for family members or other significant persons
4. Coordinated discharge planning
5. Follow-up after hospital discharge
6. Access to community resources
7. Evaluation of the patient's physical, psychological, developmental and vocational status
8. Planning for future rehabilitative and reconstructive needs

Burn Prevention Program

The burn unit should participate regularly in public burn awareness programs.

Research Program

The burn unit should participate in basic, clinical, or health sciences research.

Allograft Skin Bank Program

The burn unit is responsible for smooth functioning of allograft skin bank program including record of donors and recipients and graft utilization.

FUNCTIONING OF BURN UNIT

A burn unit of above capacity needs 1 burn unit director and minimum 2 burn surgeons besides 4 surgical residents and nursing staff as indicated. The duty roster of burn surgeons should be planned to ensure availability of one surgeon at all 24 hours. One surgical resident should be in the ICU area all the time and one more should be available in the campus at any time. For management of mass casualty situation, enrolment of all the surgical residents should be planned.

The burn team consists of surgeons, nurses, helper staff and therapists. Team work is the key to successful functioning of a burn unit. Co-ordinated efforts are also crucial for performing all the above activities. The burn unit director with leadership qualities has to nurture the burn team. The team members need guidance, direction and encouragement for upgrading their performance. Acknowledging the contribution of the team members, encouraging them to discuss their views and problems, providing psychological support during stressful periods, helps in building a cohesive team that can achieve impressive results even with scanty resources. Freedom to think and express can lead to new developments. Participation in training programs and scientific forums helps in boosting up self esteem and desire to excel. Each member should feel proud of being part of the burn team. This harmony generates sustained self motivation which can produce superior results.

Interaction Schedule

Besides daily interaction between the burn surgeon and the patient, following is the suggested schedule of interaction between the burn unit director and members of the burn team and burn team and patients and their relatives.

Schedule of minimum frequency of patient care activities:

1. Wound inspection by burn surgeon – once daily
2. Communication between burn surgeon and relatives – once daily
3. Nutrition compliance by nutritionist – once daily
4. Physiotherapy and respiratory therapy by physiotherapist– twice a day
5. Splinting and positioning by occupational therapist – once daily
6. Counseling session with clinical psychologist – twice weekly
7. Session with social worker – once weekly
8. Interaction between nurse in-charge and relatives about participation of relatives – once daily
9. Counseling by occupational therapist to all the patients and relatives about scar management – twice weekly.

Schedule of interaction between director and burn team:

1. Director and nurse-in-charge – once weekly
2. Director and nutritionist, physical therapist, occupational therapist – once weekly.
3. Director and patient's relatives – once weekly minimum.
4. Director and microbiologist – once monthly.
5. Un-announced visit by the director to assess adherence to burn unit protocol – once weekly
6. Un-announced visit to the burn unit by the microbiologist to evaluate adherence to infection control protocol – once weekly.

Funding

Burn care entails significant expenditure due to the need for intensive care, prolonged hospital stay, multiple procedures, expensive medications, long rehabilitation program and high staff-to-patient ratio. Adequate budgetary provisions should be made by the administration to meet the exigencies.

As Curreri[5] said, the burn unit succeeds or fails on the basis of managerial capability. Managers are responsible for accumulating resources, educating personnel, maintaining morale and integrating the burn unit with the rest of the hospital. Moreover, it has become apparent that burn unit managers must become financial managers as well, if we are to: 1) maintain reasonable resources for the conduct of research; 2) participate in a variety of health delivery systems; 3) curtail health delivery costs; 4) maintain the current excellence of clinical care in an efficient manner; and 5) guarantee access of patients to the burn care delivery system at a cost that the country can afford.

It is the responsibility of the Director of the burn unit to garner funds needed for smooth functioning of burn unit and for conducting different activities. Procurement and maintenance of necessary equipment depends on the ability of the director to convince the management of the hospital and on the level of commitment of the management. In the author's experience, funding can be obtained from philanthropic individuals, organizations and industry, for research and educational programs as well as for equipments. Besides the high quality of work; sincerity, commitment and transparency in utilization of funds are excellent advocates. The author truly believes that "If you have the purpose, means will follow".

SUMMARY

A burn unit is only as good as its staff.[6] Trained experienced nurses, therapists and surgeons accustomed to dealing with large open wounds are a major requisite for the care of burned patients who have a multisystem disease. The field of burn care demands high level of commitment, consistent excellent quality performance and ability to generate enthusiasm sometimes in the face of all adversities.

Burn unit's physical spaces and mechanical systems are designed to reduce infection, promote patient comfort, and improve staff efficiency and morale.[7] Patient and staff are the focal point of a burn unit and should continue to remain so for encouraging outcome.

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Chapter

39

Burn Injury Management in Disasters: Indian Perspective

Ravindra Tah

INTRODUCTION

Disasters: Definition and Impact

The Webster's Dictionary defines a disaster as 'a grave occurrence having ruinous results'. The World Health Organization (WHO) defines disaster as 'any occurrence that causes damage, economic destruction, loss of human life and deterioration of health services on a scale sufficient to warrant an extraordinary response from outside the affected community or area'.

The social and economic progress achieved over decades of initiatives by the people and advances in health can be significantly degraded by disasters.

Be it an act of God or act of Man, a mind-boggling spectrum of disasters wreck havoc in the Indian subcontinent.

The disturbing fact is that even in a region like South Asia, where poverty, deprivation and death due to disasters is a very common feature of life, India remains the worst-affected country. In fact, the frequency of all categories of disasters, varying from epidemics to road accidents, fires and perennial droughts and floods, increasing act of senseless terrorism has resulted in a multifold growth of injuries, disabilities, diseases and deaths, disrupting life-supporting systems and adding to the ill health, social and economic burden of already impoverished people.

Between 1988 and 1997, disasters killed on an average 5,116 people and affected 24.79 million every year in India. In 1998, 9,846 people died and 34.11 million people were affected by disasters.[4] Experience and study tells us that the actual figures greatly exceed the documented ones. The cities are surrounded by industrial areas which have stocks of inflammable materials like inflammable chemicals, gas storage fuel for furnaces and power generators, acrylic, woolens, textile yarn and fabrics, etc. The towns also have huge stocks of gadgets, electrical instruments not maintained or scrutinized for safety. The extensive electrification is being done haphazardly which is prone to fire accidents due to short circuits.

The National Building Code of India lays down a set of minimum provisions to which the buildings should be designed to protect the safety of the public. The regulations can be adopted immediately or enacted for use by various departments, municipal administrations and public bodies. These guidelines become mandatory provisions once the state governments adopt the code through legislation. Many states like Tamil Nadu, Karnataka, etc. have passed fire service acts to ensure fire safety of buildings.

Through this act, the fire services are empowered to issue Fire License/No Objection Certificate to building owners which enables them to get clearance for occupation from Metropolitan Development Authority/City Municipal Corporation etc. The fire license is issued based on the adherence to National Building Code provisions with respect to fire safety. In case of industrial buildings, the requirements of factories rules, Oil Industry Safety Directorate guidelines should also be complied with additionally, wherever applicable.

Despite the above mandatory requirements, non-implementation of fire safety in all buildings, residential as well as commercial, from the stage of planning is totally missing. Fires keep on happening frequently in less hazardous non industrial buildings in India.

Unregulated erection of temporary structures commonly known as "Shamianas" (make-shift tents) is done in India for all kind of functions from marriage to award giving ceremonies, including government functions. They extensively use inflammable material with loose, exposed and temporary electrical wirings making them tinder boxes which go up in flames very easily.

A review of fire safety in India reveals that the available fire services are showing signs of "deficiencies in terms of manpower and infrastructure". Instead of growing and modernizing itself to ensure the safety of life and property, the usual fire services have depleted or become deficient for the growing population. We have, beyond doubt, made the world a dangerous place to live in.

Simply defined, a disaster occurs whenever the need exceeds resources. India does not have a very well developed health infrastructure and has skewed distribution of available resources. There are a few fully equipped and staffed urban centers especially for burns injury. Therefore, any fire accident or act of terrorism resulting in large number of burn injury victims becomes a disaster. Worldwide, such a surge in burn injury causes significant morbidity and mortality. Developed and undeveloped health system determines the response and humanitarian action. India remains a resource poor country where public health system has not paid any emphasis on special development of facilities. Burns cause significant morbidity and mortality and their prompt and swift management can reduce death and disability and therefore, the need to have a plan for burn injury management in burn disaster. Lessons learnt from tackling various disasters have led to some improvement of public systems and realization of organized management for burn injuries. Preparation for burns disaster will help in improving and establishing burn care units. Therefore, every burns unit should prepare a burns disaster plan and it must be integrated with disaster planning of the emergency department and trauma care unit of the hospital. If possible, it should develop pre-hospital system or get linked with emergency pre-hospital system. It should be an integral part of the state and national disaster management plans.

The study and analysis of factors that cause a disaster, the characteristics that shape its evolution, the effects on the population and the natural environment, the instruments that can mitigate their effects and the various ways of re-establishing the optimal living conditions of the persons and communities involved have led to the creation of the new science of Disastrology. It studies disasters from all points of view and establishes guidelines for their management.

"Disaster medicine" considers the health aspects of disasters, in particular the study and collaborative application of the various health disciplines involved, i.e. from pediatrics, epidemiology, communicable disease, nutrition, public health, emergency surgery, social medicine, community care, humanitarian relief and international health, to the prevention, immediate response and rehabilitation of the health problems arising from disaster, in co-operation with other disciplines involved in comprehensive disaster management. These approaches have led to the scientific elaboration of disaster planning and response. This has been gradually transformed from a combination of ad hoc and humane actions for the stricken persons into an efficient managerial system throughout all the phases and aspects of the disaster, from prediction and prevention to preparedness, immediate medical response, assistance and rehabilitation.

Masellis described disaster as a serious and immediate threat to public health. He divided management and the rescue operations for i) a fire disaster or thermal agent and ii) a burn disaster.

Fire Disasters

It is a sudden vast ecological breakdown due to fire between man and his environment and needs extraordinary efforts to control this breakdown. This may not always result in a very large number of burn victims. Californian forest fires and Australian bush fires are the typical examples. Although a fire disaster need not necessarily reach catastrophic proportions, it will present some of the characteristic aspects of a disaster because of the highly destructive action of fire and the considerable number of victims. The surviving casualties will have mainly serious and extensive burns requiring immediate rescue procedures that cannot always be provided by local resources.

A fire of vast proportions can, moreover, cause damage to the surrounding environment by the massive production of heat and the emanation of burn gases and fumes. Smoke and gas, because of their suffocating action and their direct action on the airways, represent other specific danger elements. The danger of smoke and gas is generally underestimated by the population.

Burn Disasters

It can be defined as the overall effect of the massive action of a known thermal agent on living beings. Burn disaster can be either part of a natural disaster like lightening, forest or bush fires, earthquake, volcanoes which are rare or man made as airplane crash, building fires, explosions, fires in inflammable material industry, during transport of inflammable material or thermonuclear. A burn disaster is characterized by a high number of fatalities and of seriously burned patients with a high potential for mortality and disability. Its extent may be aggravated if appropriate rescue operations are delayed. Some decisive factors involved here are the type of causal agent, the type of pathology caused, the overall characteristics of the harmful action of the thermal agent, the immediate evaluation of its gravity in relation to emergency care and the modalities of rescue operations.

In burn disaster, two concepts are, therefore, involved, viz. the pathological condition, i.e. extensive burns and the high number of persons injured. Its extent depends on the potentially large number of fatalities which is related to the considerably large number of persons involved, the seriousness of their condition and above all, the early initiation of emergency therapy. Advances in science and technology have increased the risk of accidents and transfer of energy to nearby population. We are confronted with possibility of use of harnessed powerful sources of energy and their accidental or malicious use to unleash the magnitude of destruction which can be appalling and shocking as in the case of World Trade Centre terrorist attack. Such disaster terrorism and shift in war strategies have contributed to startling number of burn injuries or burn disasters. Therefore, a burn disaster is essentially a medical disaster for communities and the state and infrequently for the countries. It can be defined as any catastrophic event in which number of burn victim exceed capacity of local burn center(s) to provide optimal burn care. To tackle this disaster, one needs preparedness, planning, resource allocation, communication and delegation of duties.

It can become a "humanitarian emergency" when the social, economic and the medical infrastructure is disrupted or destroyed and more and more victims of burn injury are generated which cannot be dealt with the already overburdened or poorly efficient and depleted medical systems. Such, on going humanitarian crisis exists today in Iraq, Sudan and Afghanistan because even small number of burn injuries constitutes a big disaster there.

What constitutes a burn disaster in terms of magnitude, number and extent of burns is not defined. Few burn casualties may be a disaster in India in far flung places without medical facilities while a comparatively larger number of burn injuries may be dealt with satisfactorily in large cities. In USA, more than five victims of burn injury sustaining more than 20% TBSA burn is a disaster and enough to trigger disaster response, while any fire incident causing more than 25 or more deaths is a major disaster (Masellis). As said earlier, if the local facility is overwhelmed by the number of burn injuries, then it is a disaster.

Most of the burn disaster are contained and local and rarely can be catastrophic as in case of war or large explosion nuclear leak. Study of epidemiology of burn disasters makes it unlikely. It is expected that the planning for general management of disasters will cope with such rare eventuality. But even in countries which have such large scale disaster plans these plans remain untested. From the burn physician's point of view, planning would essentially focus on medical treatment and intervention in case of large burn injury disaster. The most important aspect from public health planning would be to support medical infrastructure and develop burn care units to be operational in normal times.

EPIDEMIOLOGY OF BURNS DISASTERS IN INDIA

The greatest fire tragedy in India was the Bombay Docks explosion of April 14, 1944. A fire occurred in a ship called "Fort Strikine" in the Victoria Docks. The ship was carrying ammunition and explosives. A series of several explosions occurred, destroying about 14 ships. That day 336 people died and 1,040 were injured. The explosions were recorded over 1,000 miles away at Shimla. The total loss of property according to the then calculations was approximately rupees 100 crores. This was the worst explosion in the history of fire services in India which destroyed buildings in over 100 acres of Bombay Docks area. The death toll in the catastrophe was estimated at about 500, including 66 gallant officers and men of the Bombay Fire Brigade. Those gallant officers and men laid down their lives in the cause of safety, welfare and prosperity of the community.

According to a conservative estimate of the Home Ministry, 4,000 human lives were lost along with the loss of property worth Rs. 588 crores due to fires in 1997. The actual figures may be much more, as the information collected was not exhaustive (Information Bureau, Govt. of India). Following are the details of major burn disaster in India since 1990.

April 16, 1990: Seventy people were burnt to death as a shuttle train catches fire near Patna.

Oct 10, 1990: Forty persons were killed in a fire in a train near Cherpalli in Andhra Pradesh.

Dec 23, 1995: Four hundred and forty-two people, majority of them children, were burnt to death at the Rajiv Marriage Palace in Dabwali town in Haryana. During the function of a local School there, the tent put up by the marriage palace, which had no permission to do so and flouted all fire safety norms, caught fire and collapsed over 1,000 people.

June 13, 1997: 59 people die of asphyxia in Uphaar cinema hall fire, which broke out during screening of Hindi movie '*Border*'. Over hundred persons received injuries in the subsequent stampede.

May 10, 2003, fire in a hosiery factory, godown in Khud Mohalla, Ludhiana claimed 20 lives besides leaving around 100 injured.

February 27, 2002: 58 persons, including 25 women and 15 children charred to death in the S-6 coach of the train

May 15, 2003: A burst stove caused a devastating fire that swept through a speeding passenger train in Punjab, killing 40 people and injuring more than 50.

Jan 23, 2004: 59 persons killed and injuring 60 people in a short circuit ignited fire in a marriage hall in Srirangam, a famous Hindu temple town 200 miles south of Chennai, Tamil Nadu

July 2004: 94 children perished and 100 injured in a devastating school fire, Khubhkonam Tamil Nadu.

April 10, 2006: Major fire killed at least 50 people, 115 people were injured in a blaze at a consumer fair in the industrial city of Meerut, Uttar Pradesh, 70 km from New Delhi.

Dec 10, 2008: An over loaded bus carrying 100 people broke down and caught fire. 60 people killed due to fire and 40 severely burnt.

Increasing incidents of terrorism involving explosions in recent times have resulted in multiple system trauma along with burn injuries.

The year 2008 claimed more than 210 lives and left more than 600 injured due to terrorist attacks in Jaipur (May 13th), Ahmedabad (July 26th), Delhi (September 13th) and Guwahati (Oct 30th). The worst terrorist attack on India occurred on 26th November, 2008 and left 182 dead and more than 300 injured and torched several five star hotels.

In India, during Diwali on 28th October 2008, more than 1000 fires were reported from all over country and firecrackers, most of them unsafe, worth rupees 600 crores were burst. There must be more than 5,000 injuries and at least 100 of them very serious. The fire cracker industry in India is 1,000 crores and as mentioned 600 crores worth crackers are spent around Diwali alone. The fire cracker industry is becoming more and more unregulated and is experimenting with newer and dangerous combinations to compete with Chinese firecracker industry. We create and live this disaster every year.

We do not have records of burn injuries in India. It is estimated by extrapolation of population figures that there are 7, 00,000 major burn injuries every year and about 1, 20,000 die of burns. We do not have adequate facilities in terms of bed and personnel to provide them with optimal care. So, there is an on going burn disaster in India.

VULNERABILITY ASSESSMENT AND RISK ANALYSIS

Risk analysis and vulnerability assessment involves the creation of a scenario for each potential disaster in terms of exposure, loss of life, damage to property and the geographic distribution of each hazard. Any monitoring facility, regulatory regime and counter measures available for each disaster are accounted for in this analysis.

Mass casualties from burns occur when fire and/or explosions interface with large numbers of people (Figs 39.1 to 39.3). In this context, the most likely hazards identified for India are fires, explosions, transport, train accidents, inflammable material trucks and tankers, mass gatherings especially in temporary structures, high-density urban dwellings, offshore oilrigs and acts of civil disturbance and terrorism. The recent fire disasters show that in indoor fires where the victims are trapped, immediate morbidity and the

Fig. 39.1: Historical picture depicting a major fire

Fig 39.2: Firemen in action during a fire in a high rise building

Fig 39.3: Seen of the dhabwali fire disaster

very high rate of death was due to hypoxia (inhalation injuries). Intoxication due to carbon monoxide poisoning and other toxic fumes led to inability and incapacity to react and escape and therefore, high indoor death rates as in Uphaar cinema fire tragedy.

Natural disasters such as rural fires and earthquakes fires are rare in India. In recent years, shift in terrorism to urban areas using unconventional ways to wrought destruction through explosion and fire as in the cases like 9/11World Trade Center in New York (2001), Bali (2002) and Madrid (2004). All three events highlight the fact that many patients suffered significant burn injuries as well as other multiple system traumas. Indeed, in the Bali bombings, 62 patients were admitted across Australia, occupying all adult burn beds. This extra patient surge occurred on the background of normal operations where severe burn patients from other incidents also required care. As India is becoming the most common target of international terrorism with mass casualty burn disasters occurring with catastrophic consequences, it is important to have formal plans at both a State/Territory and national level.

Historical analysis of terrorist events, where improvised explosive devices (IEDs) have been used, consistently demonstrates that up to 10-15% of the total live casualties have severe burn injury and other multiple trauma injuries requiring critical care/tertiary level burn services.

On the basis of scenario planning and historical precedent, in a worst case scenario, with 2,000 live casualties, India will have to manage a surge of up to 300 severe burn injured patients in case of the worst explosive attack. This will significantly challenge existing systems.

Burns injuries in a disaster or burn injury disaster can not be viewed only as a medical emergency and their management cannot be done in isolation. With the increasing awareness about scientific ways to manage a disaster, it becomes imperative that management of burn injury is an integral part of overall disaster management.

The limited scope of interpreting a burn injury disaster from hospital or health care point of view has to change to holistic disaster planning.

Therefore, all burns disaster plans have to be part of the comprehensive, multi-hazard disaster management plans, emphasizing disaster management response, disaster awareness and education.

APPROACH TO DISASTER MANAGEMENT IN INDIA

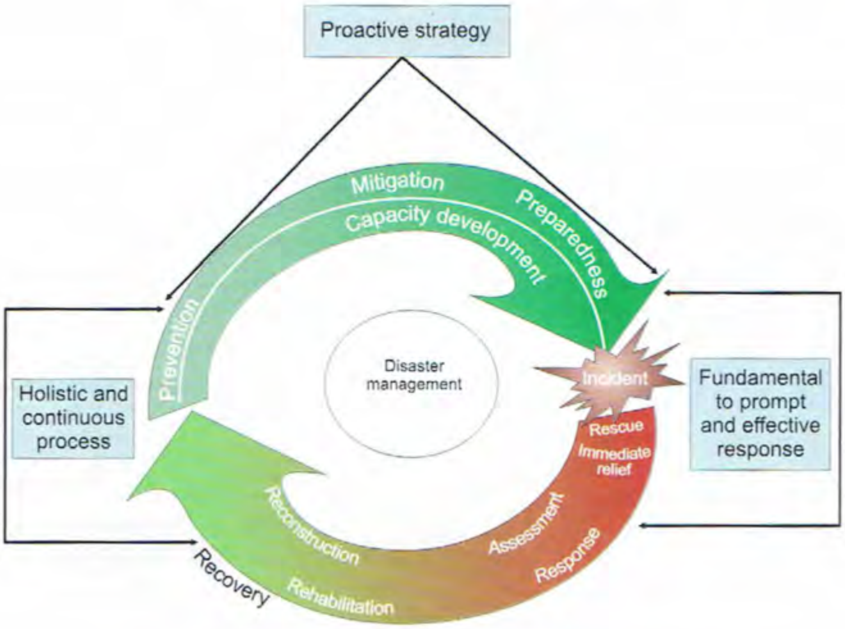
The increasing frequency and fury of natural disasters has shaken the policy makers of India and a high power committee has been established to address the multiple facets of natural and man made disasters in India.

Till recently, the approach to disaster management has been reactive and relief centric. A paradigm shift has now taken place at the national level from the relief centric syndrome to holistic and integrated approach with emphasis on prevention, mitigation and preparedness. These efforts are aimed to conserve developmental gains as also to minimize loss of lives, livelihood and property.

A typical Disaster Management continuum as shown below, comprising of six elements i.e., Prevention, Mitigation and Preparedness in pre-disaster phase and Response, Rehabilitation and Reconstruction in post-disaster phase, defines the complete approach to Disaster Management (Flow chart 39.1).

Government of India (GOI), in recognition of the importance of Disaster Management as a national priority,

Flow chart 39.1: Disaster management continuum



set up a High-Powered Committee (HPC) in August 1999 and also a national committee after the Gujarat earthquake, for making recommendations on the preparation of Disaster Management plans and suggestions for effective mitigation mechanisms. The Tenth Five-Year Plan Document also had, for the first time, a detailed chapter on Disaster Management. Similarly, the Twelfth Finance Commission was also mandated to review the financial arrangements for Disaster Management.

On 23 December 2005, the Government of India enacted the Disaster Management Act (Flow chart 39.2), which envisaged the creation of the National Disaster Management Authority (NDMA), headed by the Prime Minister and State Disaster Management Authorities (SDMAs) headed by respective Chief Ministers, to spearhead and implement a holistic and integrated approach to Disaster Management in the country.

National Institute of Disaster Management (NIDM)

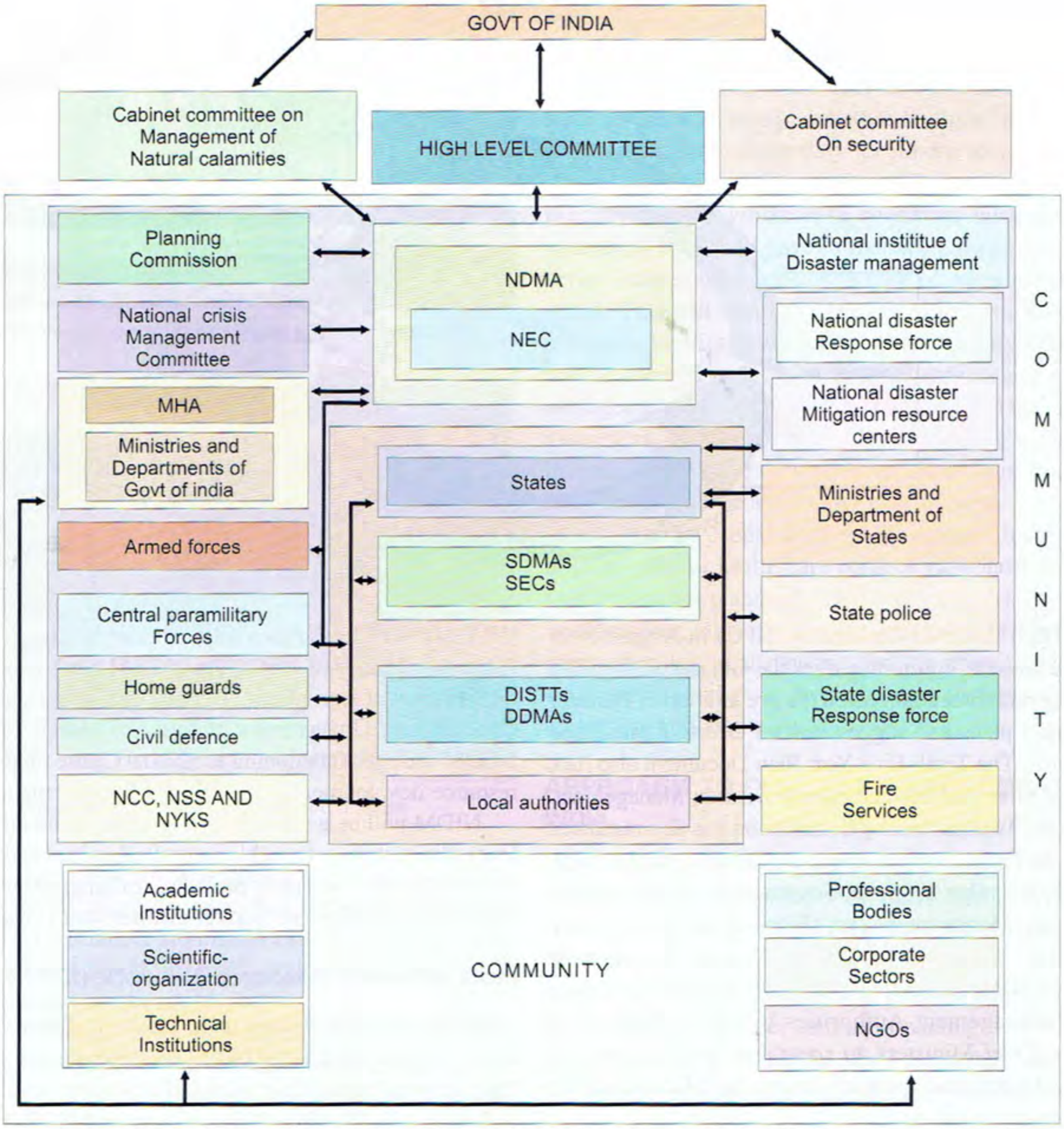
Under the NDMA and through disaster management act, a National Institute of Disaster Management (NIDM) was created. It is a premier national organization working for human resource development at national level in the area of disaster mitigation and management. The NIDM came into existence on October 16, 2003 by a Government of India order upgrading the National Centre for Disaster Management (NCDM), which was located at Indian Institute of Public Administration, New Delhi. The NCDM was established by the Ministry of Agriculture, Department of Agriculture and Cooperation, Government of India in March 1995. The NCDM had been functioning as a Nodal Centre for the human resource development in the area of disaster management.

NIDM will be gearing up the national, state and district level administration to tackle natural calamities and will also be coordinating research projects, training programs and will build a database on natural disasters with case studies.

India Disaster Resource Network (IDRN)

Under the national disaster plan, Ministry of Home Affairs, Govt. of India, established India Disaster Resource Network. This is the net work of net works. IDRN portal was launched in September 2003 in collaboration with UNDP. Technical support was extended by UNDP to systematically formulate the resource inventory through an organized information system designed to collate and assimilate information from authorities in 563 district and 35 State/Union territories. The on-line inventory clearly lists out sequential details on specific equipments, types, their functions performed, human expertise in search and rescue operations and contact details of expertise and suppliers for prompt mobilization. IDRN was designed as a web enabled information system to provided updated inventory of related items every quarter,

Flow Chart 39.2: National Disaster Management Structure


GOVT OF INDIA
Cabinet committee on Management of Natural calamities
HIGH LEVEL COMMITTEE
Cabinet committee On security
Planning Commission
National crisis Management Committee
MHA
Ministries and Departments of Govt of india
Armed forces
Central paramilitary Forces
Home guards
Civil defence
NCC, NSS AND NYKS
NDMA
NEC
States
SDMAs SECs
DISTTs DDMAs
Local authorities
National instititue of Disaster management
National disaster Response force
National disaster Mitigation resource centers
Ministries and Department of States
State police
State disaster Response force
Fire Services
COMMUNITY
Academic Institutions
Scientific-organization
Technical Institutions
COMMUNITY
Professional Bodies
Corporate Sectors
NGOs

Notes: 1. This diagram reflects interacitve linkages for synergised management of disasters and not a hierarchical structure.
2. Backward and forward linkages, especially at the fuctional level, are with a view to optimise efficiency.
3. Participation of the community is a crucial factor.

entered at two levels, i.e. at district and state level. The portal has been hosted to the central server at National Information Centre (NIC), who is providing technical support to Ministry of Home Affairs. The portal has an online dual accessible interface system for users from state disaster management/relief and rehabilitation department officials (Relief Commissioners), district administrators (District Magistrate/Collectors) and other state and district nodal officers, corporate members and public who can browse the portal. The user can obtain catalogued information as well as query based data and list of resources available at district and state level. The on line inventory user manual and details can be accessed at http://www.idrn.gov.in

On the IDRN, nation wide availability of beds for burn injury can be listed along with trauma units and their surge capacity. National Academy of Burns-India (NABI) should provide authentic information after getting all the information from the public and private hospitals on IRDN to be used at the time of disasters. The list of burn care physicians should also be provided area-wise with their contact information for use during disaster.

Burn Disaster Response Team (BDRT)

At present no plan for burn disaster response team has been made in disaster planning in India a whole or in any other state. A burns disaster team can be raised in larger institutes, e.g., Safdarjung Hospital in New Delhi, as they are the repository of large number of trained burn care experts. At least one team should be readied with personnel, equipment and resources to be dispatched at short notice from the department of burns and plastic surgery at Safdarjung Hospital, which is directly under Central Government. It will help dispatching such team promptly as sending an international and interstate humanitarian mission requires government permission.

Burn care is not optimal in all countries and even in India only very few centers provide desired optimal care. Therefore, there is a need to raise burn disaster response teams. Government of India or NABI and (Burn Association of India (BAI) should prepare a plan to constitute such burn disaster response teams and offer these teams for national needs in areas of deficient expertise or for international humanitarian missions.

BDRT can travel at short notice to the disaster site and carry out effective primary triage, first aid and execute safe transport. BDRT should have a designated leader and team members. The vehicles and resources should be planned and earmarked.

IDRN can also enlist information about individuals and agencies who can volunteer at the time of disaster and humanitarian crisis.

To build a safer and disaster resilient India by developing a holistic, pro-active, multi-disaster and technology-driven strategy for disaster management, collective efforts of all Governmental Agencies and Non-Governmental Organizations are required.

National Disaster Response Force (NDRF)

The Concept: The Disaster Management Act has mandated the constitution of a Specialist Response Force to a threatening disaster situation or a disaster. This Force will function under the National Disaster Management Authority which has been vested with its control, direction and general superintendence. This will be a multi-disciplinary, multi-skilled, high-tech force for all types of disasters capable of insertion by air, sea and land. All the eight battalions are to be equipped and trained for all natural disasters including four battalions in combating nuclear, biological and chemical disasters (Fig. 39.4).

Present Organization: Presently this Force is constituted of eight battalions, two each from the Border Security Force (BSF), Central Reserve Police Force (CRPF), Central Industrial Security Force (CISF) and Indo Tibetan Border Police (ITBP). Each battalion will provide 18 self-contained specialist search and rescue teams of 45 personnel each including engineers, technicians, electricians, dog squads and medics/paramedics. The total strength of each battalion will be approximately 1,158.

Deployment: These NDRF battalions are located at nine different locations in the country based on the vulnerability profile to cut down the response time for their deployment. During the preparedness period/in a threatening disaster situation, proactive deployment of these forces will be carried out by the NDMA in consultation with state authorities.

There is an urgent need to establish national burn injury response team which can be done as mentioned earlier at few large institutes in the country. One of the ideal centers to constitute such a team will be Safdarjung Hospital, New Delhi as it is repository of largest number of burn specialists and paramedical and the trainees directly under Government of India.

National Disaster Mitigation Resource Centers (NDMRC)

The NDMRCs will be co-located with the NDRF battalions. These will also serve as repositories for NDMR bricks of relief stores for 25,000 affected people, in each of the nine locations. These will cater to the emergent requirements especially for the first 72-96 hours. At Kolkata and Chandigarh, additional bricks of stores for 50,000 people each will be kept for high altitude areas. These stores will supplement the reserves maintained by the respective States. In addition, these centers will assist in running mock drills and capacity development programs. During disasters, they will act as facilitators to the States/Union Territories in deployment of central resources and provide much needed additional link to the center.

NATIONAL DISASTER MANAGEMENT GUIDELINES-MEDICAL PREPAREDNESS AND MASS CASUALTIES MANAGEMENT (NDMG-MPMCM)

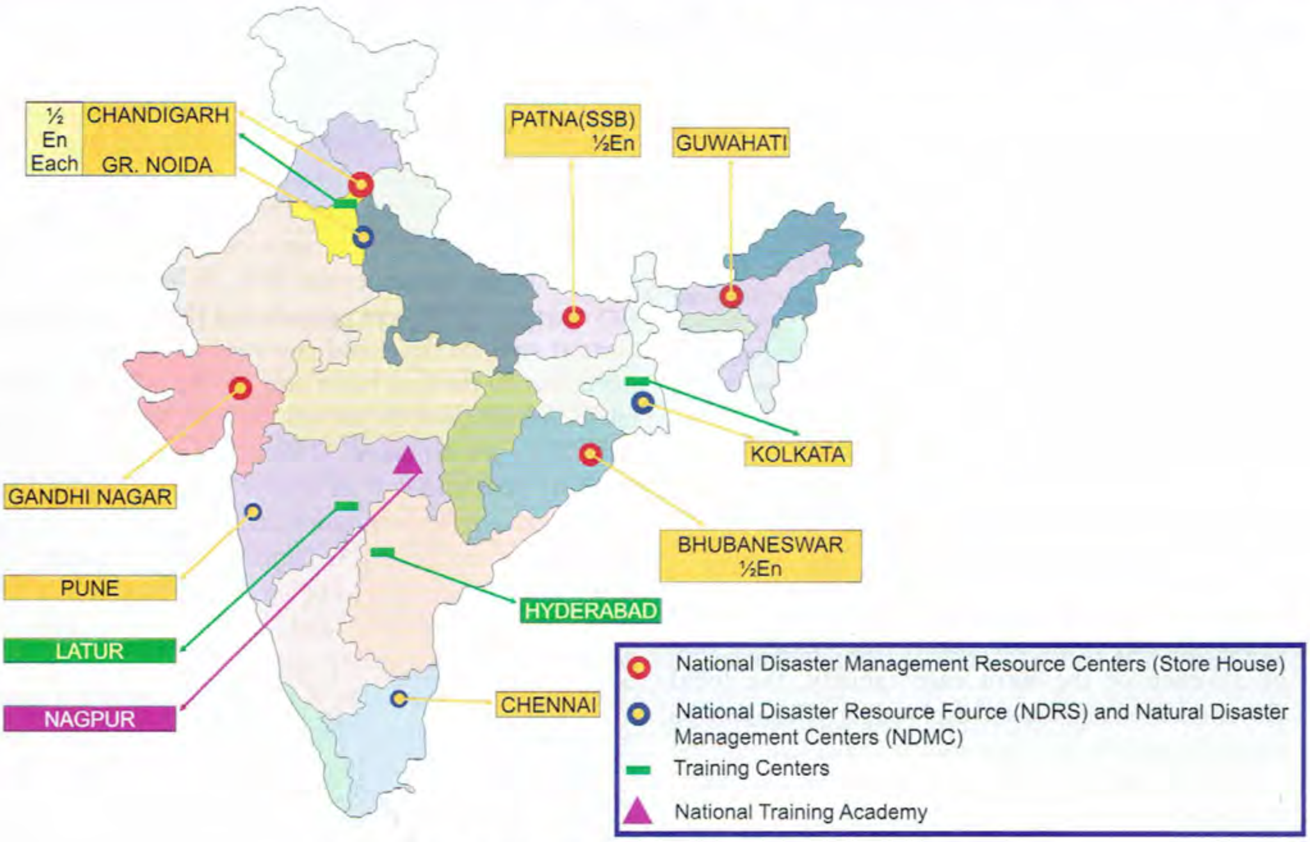
Medical Management of Mass Casualties is based upon 'all hazard' concept where practically every Ministry/ Department will be playing a role directly or indirectly with nodal ministry as Ministry of Health and Family Welfare, Government of India. As per the National Disaster Management Act, 2005, National Disaster Management Authority (NDMA) is required to prepare Guidelines, based on which, the Nodal Ministry will prepare a detailed Action Plan for the better and effective medical management of mass casualties. In view of evolving such Guidelines, a National workshop on Medical Preparedness and Mass Casualties Management was convened by NDMA in August, 2006 and various experts from the Center, State Governments and NGOs participated. In this workshop, the present status of the Medical Preparedness in the country was discussed and future course of action was set up. It was decided to articulate the MPMCM guidelines through a document called National Disaster Management Guidelines-Medical Preparedness and Mass Casualties Management (NDMG-MPMCM). A Core Group of the experts from the various fields related to medical management was constituted under a Chairman to assist NDMA in preparing National Guidelines for MPMCM. The core group has met for deliberations and has yet to finalize its recommendations. NDMA has observed some critical gaps in managing mass casualties and issued fresh guidelines for the disaster and need to adopt multi-disciplinary and multi-sectoral approach for prevention/mitigation strategies so as to develop capacities to improve response. It envisages strengthening of about 200 hospitals from basic trauma care to advanced tertiary care, all networked with pre-hospital care ambulances to provide care during transit and hospitalize within the golden hour.

Essentially, apex bodies of central government are trying to associate other professionals. Surprisingly no associates of medical fraternity, especially the burns care bodies like NABI (National Academy Burns-India) or burns care experts have been nominated. There is an urgent need for NABI to take this issue with MP-MCM group. A model integrated plan applicable to all districts of India for management of burn injury mass casualty should be submitted. Making plans in isolation will not be effective as NDMA is planning disaster management to the level of districts through state government.

The Section 23 of the Disaster Management Act 2005 provides that there shall be a disaster management plan for every state. It outlines the broad coverage of the plan as well as the requirements of consultation in the preparation of the state plans. It also provides for annual review and updating of the state plan, and enjoins upon the state governments to make provisions for financing the activities to be carried out under the state plans. It provides for the departments of the state governments to draw up their own plans in accordance with the state plan. The state plans shall be prepared by the State Executive Committee (SEC) in conformity with the guidelines to be issued on related matters by the SDMA having regard to the guidelines laid down in this regard by the NDMA and after such consultation with local and district authorities and the people's representatives as the SEC may deem fit.

With assistance of UNDP and USAID under the SDMP the states have been assisted to draw district and block level DMP through the Panchayti raj institution. The DMP has been prepared for 8643 villages, 1046 Gram Panchayats, 1818 blocks and 82 districts. More than 29000 elected representatives have already been trained besides volunteers at village level who are being trained. Besides this, 18,000 government officials, 865 engineers, 425 architects, 600 master trainers and 1200 teachers have already been trained.

Twenty nine states have disaster management facilitates and plans finalized. 28 of them have created training institutes. State of Gujarat, Maharashtra, Assam and Bihar have finalized their disaster plans and have published them.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HYDERABAD
NAGPUR
CHENNAI
National Disaster Management Resource Centers (Store House)
National Disaster Resource Fource (NDRS) and Natural Disaster Management Centers (NDMC)
Training Centers
National Training Academy

Fig. 39.4: National disaster response battalions and training centers

In an effort to create awareness and plan for future management of disasters, disaster management has been introduced as subject in class eighth and ninth by Central Board of Secondary Education (CBSE), New Delhi.

In major cities (with a population exceeding10 lakhs), municipal corporations have a large administrative system including departments like engineering, public health and revenue and sometimes fire services. These should provide a good mechanism for coordinated response in case of any crisis/disaster. Therefore, management of major cities should be planned as a unit. In metropolitan cities it is also advisable to make the urban metropolitan government directly responsible for DM.

The Medical Preparedness and Mass Casualty Management

The following points must be reflected in the state plan:

- Listing of all hospitals and Primary Health Centers including private nursing homes and their capacities.
- Emergency casualty management plans at the local, district and state level.
- For effective management of big cities like Delhi and Mumbai, nearby medical facilities must also be built into plans.
- Availability of mobile hospitals and/or plans for acquiring the same. The state plan may also adopt the Generic Categorization of Disasters with specific plans at the state level by various departments to handle different disasters.

Levels of Disaster

The levels of disasters have been categorized and disseminated as L0, L1, L2 and L3, based on the ability of various authorities to deal with them. Various color codes relating to the level of alerts have also been assimilated. L0-denotes normal times which are expected to be utilized for close monitoring, documentation, prevention, mitigation and preparatory activities. This is the planning stage where plans at all levels from community to the State shall be put in place. Training on search and rescue, rehearsals, evaluation and inventory updating for response activities will be carried out during this time.

- L1-specifies disasters that can be managed at the district level. However, the state and center will remain in readiness to provide assistance, if needed.
- L2 -specifies disaster situations that may require assistance and active participation of the state and the mobilization of resources at the state level.
- L3 -disaster situations arise from large scale disasters where districts and the state may not have the capacity to respond adequately and require assistance from the central government for reinstating the state and district machinery.
- Even though there cannot be water tight compartmentalization of responsibilities between states and the centre, especially in the case of man-made disasters, the participation by Centre will normally beat the L3 level. Most of the burn disasters would fall in L-1. or L-2 types. The burn care facility in the district has to coordinate with the district disaster plan (DDP). It has to become part of the community based DDP. In case of absence of the burn care facility, the local Government and private medical facility will have to be enlisted to provide initial care and plan evacuation to the nearby burn facility.

Role of the Emergency Operations Center (EOC)

Time bound action plans must be indicated for creating facilities for environment scanning, intelligence gathering, operations and logistics management under one roof for holistic and efficient management of disasters at the state and district level. Arrangements must be made and procedures must be laid down for manning the EOC on 24 × 7 × 365 basis. The plan must also cover in detail the indigenized Incident Command System at the state, district and local body levels.

The disaster specific plans for mining, oil spills, biological, chemical and nuclear has been made by some States but no burn disaster specific plan has been made by any state or city administration. It is felt that bodies like NABI, with local burns care facilities, should develop burn disaster plans taking into consideration the local facilities, geographical conditions etc. A model burn disaster plan for cities and rural area can be prepared and submitted to NDMA coordinating the activities of various health disciplines to prevent disasters, provide an immediate response to a disaster and help in rehabilitation of victims.

BURN INJURY SCENE IN INDIA

In India, specialized burns care is available for the last 50 years but gloom prevails over this particular discipline, marked by alarming morbidity and mortality. On an average, burns centre of a public hospital is able to save patients with burns below 40 per cent. Those having burns close to 50% have 50% chances of survival and very few cases survive having burns beyond 60%. In West Bengal, the survival rate of a 50% burn is a dismal 25%. This is in stark contrast with the developed countries where patients with more than 70 per cent burns also survive and the survival rate of patients with 50 per cent burn is from 75 to 90 per cent. There are estimated 32 burns units India which are woefully inadequate. With around 7, 00,000 major burn victims needing hospitalization and 1, 20,000 succumbing to the injury, the meager 32 burns units in the country (of which only 19 are said to be up to the mark with a low mortality and morbidity rate), are not able to meet the demands and render quality care.

India with a population of more than one billion needs ten times more number of burns units in the country. In the US, there are about 120 burn care facilities for a population of less than three hundred million.

Burns care in government hospitals is plagued by lack of infrastructure, whether it is sophisticated equipment, availability of medicines, spacing between beds or the manpower. With burns care needing the facilities of an intensive care unit, some burns units are run without basic equipment like ventilator, pulse oximeter, infusion pump and patient weighing machine and skin graft mesher etc.

The burns unit is not something that the private sector evinces interest with problems of high infection rates, medico-legal work (with every burns case to be reported to the police and requiring evidence in the court in case of complications) and a long hospital stay.

While the chances of survival of severe burns patient can be enhanced by skin grafting, burns surgeon rue that cadaver donation of skin cannot be practiced with the non-inclusion of skin in Transplantation of Human Organs (THO) Act, 1994. Though burns surgeon did lobby to allow cadaveric donation of skin, the government had turned down the proposal on the ground that skin is a tissue and not an organ and hence cannot be included in THO Act. Live skin donation is hampered by unavailability of donors, the risks associated with contacting AIDS and the pain and trauma that the donor has to undergo.

Although there are more than 1200 plastic surgeons in India who constitute a useful resource of trained personnel in burns, most of them dedicate themselves to burns care only partially. Not more than 40 burns surgeons are exclusively dedicated to burns care. All the burns units in government hospitals are usually full. Therefore, there is hardly any surge capacity in specialized care units for burns injuries in India

SURGE CAPACITY OF HEALTH CARE UNITS

Surge capacity has been defined as the "health care system's ability to manage a sudden or rapidly progressive influx of patients within the currently available resources at a given point in time. This term entered the disaster medicine lexicon after "9/11" 2001 in America, and has become a key feature of health disaster planning since the severe acute respiratory syndrome (SARS) outbreaks, the rising threat of pandemic influenza and the Madrid and London bombings in 2004 and 2005 respectively.

Mass trauma events in urban environments create surge problems for hospitals in the first 4 hours, with the majority of casualties likely to arrive at the hospital in the first 60-90 minutes. Health systems need to be able to meet this increased demand after acute mass casualty events, with an influx of patients who may require "specialized evaluation or intervention" specially burns, including surgery for penetrating trauma. As the epidemiology of traumatic events will differ depending on the cause, any preparations to manage a mass casualty event needs to be flexible enough to deal with a range of disaster types, including infectious disease emergencies and bioterrorism, with their more insidious and later-peaking surge.

Meeting the challenges of surge capacity enhancement calls for cooperative action at the complex interface between clinical practice, health systems management, logistics, facilities management and personnel deployment.

The US Health Resources and Services Administration has set a minimum benchmark for surge capacity of 500 adult and pediatrics patients per million population for infectious disease events and at least 50 patients per million population sustaining trauma or burns in a mass casualty event. These figures have not been validated and based on other traumatic events, 100-300 patients per million may be more accurate for burns or trauma. The consensus from a series of articles on the "science of surge" in the November 2006 issue of Academic Emergency Medicine was that there is little or no quantifiable measurement for managing an acute "sudden impact" surge or the even less predictable surge resulting from infectious disease emergencies or obscure events.

Some of the measures which can be implemented in case of huge surge are:-

- Creating temporary hospital facilities within public hospitals by having caches of medical supplies and equipment available;
- Using alternative health facilities, such as operating theaters, beds and diagnostic resources in private hospitals and day procedure units for the "walking wounded" and "decanted" patients (patients already in hospital who can be transferred elsewhere to make way for disaster victims);
- Using general and private health practitioners to provide a range of care for the walking wounded and decanted patients; and
- Applying modified models of care, such as longer shifts and extended scope of practice for nurses, allied health practitioners and medical and other health students.

PREVENTION AND MITIGATION

Prevention is the most important public health tenet.

Pure disaster response alone is not sufficient and gives only tiding away and temporary results at a very high cost. Prevention and mitigation contribute to lasting improvement in safety and are essential to integrate in disaster planning.

To avoid a mass burn casualty disaster, planning from several key government areas is required in terms of legislation and strict implementation.

Industrial, high-density, urban fires—all buildings should have fire prevention strategies (sprinklers, smoke alarms, and fire egress). All residents of the buildings should be briefed on fire prevention and escape. This will be the responsibility of state operated fire services.

It has been noted that although strict laws for buildings and other structures exist, it is the poor implementation and lack of will to maintain fire safety equipment which is the root cause of failure for prevention. Most of the Indian states have enacted their building codes and fire safety acts to meet the requirement of high rise buildings.

Inspection of all buildings for their fire safety worthiness should be done on regular basis. Fire escape plans must be displayed properly. Every room of a public building should have fire escape plan displayed.

The fire departments under state governments are not well maintained and staffed. They need to have better allocation of funds to strengthen fire fighting capacities.

Although there is national fire safety institute in Nagpur but more needs to be done in the field of fire safety.

Forest fires—Although forest fires are rare in India, these fires have been reported sporadically from Himachal Pradesh in north India and Tamil Nadu and Karnataka in south India. Forest department, usually with district authorities, takes care of these fires.

Transport—All transport devices should meet the criteria for transport of hazardous materials. Responsibility: State Road and Transport authorities, Ports authorities, Commercial Aircraft Safety Authority should implement the laws strictly.

Terrorism—To prevent a mass-burn disaster through an act of terrorism, intelligence from agencies such as newly created National Intelligence Authority, state intelligence, border security forces and national body to fight terrorism is required. In the wake of terrorist attack from sea, strengthening of our coast by coast guards and navy is required.

Information should be freely available to all residents on what to do and where to go in the event of a major disaster – i.e. a page in the telephone directory.

Mitigation Strategy

The mitigation strategy and plan focuses on the long-term planning for disaster reduction. It deals with the issues of continued commitment to hazard identification and risk assessment, applied research and technology transfer, investment incentives for mitigation and leadership and co-ordination for mitigation. The mitigation strategy makes an argument for better land use management, building codes, traffic standards and health standards. These objectives are to be secured through disaster legislation, mitigation regulation and incentives for mitigation.

At the state and district levels, the same structure is followed. The Disaster Management Plan calls for the creation of a variety of facilities and resources which provide support in the nature of infrastructure and organizational requirements.

FUNDAMENTALS OF BURN DISASTER PLANNING

The burn injuries are common in mass disasters and terror attacks.

The disaster plan is the best tool for preparedness.

Burn center care is the most efficient and cost effective care of burn injuries.

Burn centers are not same as trauma care centers. Burn centers are unique national resources. NABI, National Academy of Burns-India, has the capacity to be a key component in national disaster readiness for mass casualty.

Planning requires pre-existing health and medical infrastructure and cooperation with local and state Governmental agencies. Specialized burn care facility and the team are the key around which the plan must be developed. There has to be an active encouragement to lay out plan for emergencies. All the stakeholders in burns mass casualty should be made partners. They are police, district civil authorities, fire department, emergency medical services, ambulance services, transport providers, health department, health centers, hospitals burns care units, private physician and NGOs.

The objective is to provide optimal treatment of disaster victim at the earliest. Though detailed plans through state disaster management to the level of district have been worked out, burn mass casualty management has not been included. The burn care organization and the specialized burn care units need to draw the attention of the state and national planners and get themselves included in the disaster plans. Scientific bodies like NABI should coordinate with state and national authorities.

Burn disasters are usually man-made, usually confined to local and limited geographical area. Consumption in terms of man power and material are maximum for burn injury. Even three to four patients have the capacity to strain health facility. More than 20 patients will require intervention from multiple well equipped facilities. In urban areas of India, it is possible to cope with 20 to 30 victims of burn injury. Magnitude of 100 to 200 victims will overwhelm the medical system but we are not at all prepared for stronger surge. We need to plan for large scale burn disasters, especially with increasing terrorism in recent times with trend of multiple bombing at multiple sites.

According to Masellis and Gunn, the following points should be considered when planning burn disaster plans.

a. The time interval between the accident and initiation of care must be less than two hours.
b. Triage of victims must be carried out by specialists, as specialist alone can evaluate the gravity of burn injury.
c. Inhalation and combustion of gases, fumes and hot air causing damage to airway alone can jeopardize survival.
d. Hypovolemic shock is induced rapidly and tissue hypoxia induces irreversible changes in various organs. It should be addressed immediately.
e. Burn may be associated with large wounds, fractures, electrical injuries or blast injury lesions.

f. The overall assessment should be made not only on the basis of dead but also on the number of injured and those likely to suffer disability.

g. The place where disaster occurs may not be accessible and no disaster plan to respond exists.

Of the 14 burn disaster studied by Arturson, only in San Juanico tragedy of Maxico where LPG storage tanks caught fire, more than 600 were dead with count of injured exceeding 7,000, the worst burn disaster on record in recent history, the disaster plan was in place .

Immediate care is provided by people present at the scene of the disaster, who may be survivors or passers by. These first responders are later guided by trained health care workers who arrive at the site. On site management includes:

Immediate relief and first aid: Immediate assistance, provided in first 2-3 hours by the people preferably by trained nurses, physicians and volunteers, is fundamental to prognosis of burn patients. More complex and organized relief may take 6-7 hours to reach. It may be too late for burn patient. When burn casualties exceed the local ability, a major disaster should be declared. District disaster plan will take about 20 burn patients. In case of more casualties, the state plan should be activated. All the agencies should be utilized and educated about their roles and responsibility to avoid chaos at the scene.

UTILIZATION OF MEDICAL TRIAGE

Ambulance staging with a basic aim of maximal use of resources, efficient and safe transport.

Early notification and information to hospitals.

Utilizing an existing point of entry to activate disaster plan.

TRIAGE

Sorting out and classifying casualty of war or any other mass casualty is known as triage. It is done to determine the priority and proper place for transfer. Triage is most aptly applied to burns as burn injury is to some extent quantifiable.

Triage is the cornerstone of effective burn disaster management and is done at the disaster site by staff with knowledge of burn treatment. Triage takes into consideration the total number of patients, bed availability and transportation capacity. The guiding principle is to treat those who are most likely to survive or can benefit the most from treatment. Thus, triage standards address who receives care and when care is provided or the urgency with which it is provided. Triage is performed most often by first responders. Triage begins in the field, if there is a fixed event site; however, it also occurs within care settings, such as hospitals and alternate care sites, where individual victims may present themselves for care independent of organized responses. Secondary triage also may be necessary within a facility, such as a hospital, as demands on the system grow. Several well-established standards for triage are currently in use. Triage systems include START; Jump START (a pediatric modification to START); and Sacco score system. Each system seeks to establish a small number of categories among victims that indicates the urgency with which they should be treated. Colors are often used to represent the categories—for example, red (immediate care); yellow (delayed); green or white (ambulatory and minor injuries); and black (dead and/or "expectant).

The adequacy of the triage system used depends on the nature of the event and the population affected. For example, systems such as START and Jump START are trauma-oriented and may be effective in an explosive event.

In case of burn disaster or a disaster with burn injuries, the most important aspect would be to segregate patient with only burn injury or burn injury associated with other injuries for examination and triage by burn specialist or physician trained in burn triage. If the incident is small with limited casualties, ambulance trained paramedical will carry out primary triage and transport.

Triage should be prognostic, and patients should be categorized on the basis of age, extent of burns and site of burns and presence of inhalation injury. Although there are no specified triage criteria but recommendation for transferring patients to a burns unit for satisfactory management is the guiding principle, which are as follows:

- *Group I*—Minor burns (< 10% of total surface area in children, < 20% in adults) to non-critical areas.
  Assigned to—Outpatient care, dressing, tetanus prophylaxis.
- *Group II*—Minor burns to critical sites (face, hands, genitalia)
  Assigned to—Short hospital stay, special wound care or operation.
- *Group III*—Major burns (20-60%)
  Assigned to—Admission to burn unit, intravenous resuscitation.
- *Group IV*—Extensive burns (> 60%)
  Assigned to—Lower priority for transfer.
- *Group V*—Minor burns with inhalation injury or associated injury.

Assigned to—Oxygen, intubations, transfers to intensive care unit.

The patients in groups III and V are evacuated first, followed by group IV. Group II cases are evacuated at the end. Group I cases are either discharged after first aid or asked to make their own way to the nearest primary care center. Once the surge capacity of nearest burn center is exhausted, transfer to another center. If the surge is too huge, then a trauma center serves as next level to provide treatment.

Tele-triage can be done by getting in contact with any tertiary care unit where a burn specialist can guide in triage and initial management of the patients and the process of preferable transport can be initiated. There is a need to train primary physicians and all emergency medical officers in the early management of burns and the method of triage. An organization like NABI can play a very vital role in this education.

As mentioned earlier, ideally the triage should be done at the site of accident itself or should be set up at a nearby dispensary or health unit. In India, there is no instance where the triage has been used or set up. Patients have been loaded and taken to nearby facility where some sort of triage based transfer has been done.

COMMUNICATIONS

Communication during disaster is the most vital tool. It is desirable to have on line voice communication to ambulance service to burns unit, hospital coordinator burns surgeon, trauma center individually and collectively. In some developed countries, this aspect is already operational, e.g. INFOBRUL in France, NDMS in the US, Argo in Italy. Like USA, Germany has enhanced centralized system which communicates to fire ambulance service and hospitals and police at the same time. In India, none of the states or cities has tried to develop and use communication and command centers. The communication command centers should be developed and manned by police and civil administration. It should have central data bank about ambulances evacuation, transport availability, disaster teams locally and hospital beds availability. It should be manned 24 hours by personnel trained in disaster management. The best way to keep this communication center developed is to use it on daily basis for routine distribution of patients of trauma and routine emergencies. In India, under disaster plan, district control room (DCR) and emergency operations center (EOC) are being developed. The state of Maharashtra has put Communication network comprising wireless (VHF) and satellite (VSAT) networks covering the entire state and computerized disaster management information system using geographic information system (GIS) applications.

District Control Rooms: District Control Rooms are to be established in each collectorate of the state. The control rooms serve as nodal facilities for directing operations in an emergency situation. The most important is to develop links to organization capable of mobilizing carriers for transport of patients and send prior information to the hospitals of patients it is likely to receive. The first information usually received by these center will trigger disaster management response.

Emergency Operations Center (EOC): The Emergency Operations Center is a well-equipped and modern facility located at the state government headquarters. The EOC is the hub of activity during a disaster. Its systems and procedures are designed in such a way that information can be promptly assessed and relayed to concerned parties. As the master co-ordination and control point for all counter-disaster efforts, the EOC is the center for decision-making under a unified command. It is to be linked with all district control rooms.

MANAGEMENT AT DISASTER SCENE

In an episode of disaster, the information should be passed on to police and district administration. In India, in all disasters the management is taken over by police and district civil authorities or in worst cases by the paramilitary and military forces.

Evacuation and transport: First task is to find, extricate and transport victims usually by first responders or by fire services who are professionally trained. The effort should be to recruit all those who can rescue because early rescue is the key to greater number of lives saved. The response plan should take into consideration the activation plan for explosions and industrial accidents especially in districts having petrochemical and refinery industry. If the disaster occurs in a remote area or the district has hardly any capacity to deal with large number of burn victims, then establishing of field hospital should be considered. In India, as mentioned earlier, field hospital with the disaster management team is being readied. In normal circumstances, the response is to load and go to the nearest health facility. The "load and go" evacuation system must never be used, especially in burn disasters. It causes great hold-ups in transport, a chaotic use of specialized beds and considerable risks for patients who receive resuscitatory treatment only after long delays. If possible, the primary triage should be conducted near the scene of disaster and evacuation should be prioritized so that the scarce bed capacity at the hospital is not over burdened and low priority patient may choke the system.

Modern disaster management plan should effect controlled and safe transportation of burn patient to meaningful destination. Early information and notification should be sent to potential burn facility to receive patient.

Emergency transport of burn patient is to be coordinated with rescue organization. It may be practical to transfer to the nearest medical facility for thorough assessment and life saving treatment of respiratory and airway management.

Burn injury tolerates transport best on the day of accident. Ideally all victims should be transported within 60 minutes. Late transfer is best done on third or fourth day before sepsis sets in. Mass burn casualty in urban areas must take into consideration traffic jams due to disaster. Therefore, corridors of fast transfer to hospital should be worked out before hand and if possible civil authorities should keep access to hospitals clear. Traffic jams can also affect arrival of staff to the hospital for disaster management. In India, there is no provision of air or helicopter evacuation. It is only rarely done for very important persons and in rare circumstances.

When military forces are involved, there is a need to establish regional helicopter evacuation services because it is rapid and are not hindered by chaos on the ground.

When the surge is large and in remote areas, establishing a field hospital should be considered. Field hospitals which can be flown rapidly and established are only available with army and para military forces. As mentioned earlier, the national disaster plan is building and strengthening these field hospitals with special battalions.

Burn facility surge capacity and Hospital Emergency Incident Command System (HEICS):

Burn patients are best cared in burn units and there would always be scarcity of specialized beds and expertise for the treatment of extensive burns. Traditionally, the role of burn units is to provide medical management to the patients who arrive or get admitted. With disaster planning, the extended role is to keep team ready and send them to disaster scene to help evacuation, triage and initial management, to coordinate and focus on leadership within medical component.

Burn units rarely have low occupancy. Therefore, there should be an emergency plan to shift and evacuate existing burn patients to accommodate mass burn casualties. The thumb rule should be to have at least 50% more capacity to accommodate. As the burn units are usually located in large hospitals, the hospital should develop and have Hospital Emergency Incident Command System [HEICS] in place and this should be tested for burns mass casualty. HEICS provides responsibility oriented chain of command which is charted and available with every one following case of accident or emergency. It provides for Prioritization of duties with the use of Job Action Sheets. The Job Action Sheets are position job descriptions which have a prioritized list of emergency response tasks. The Job Action Sheets also serve as reminders of the lines of reporting and promote the documentation of the incident.

- A dependable chain of command.
- Improved communication through common language.
- Flexibility.
- Prioritization of tasks.
- Organized documentation system.
- Effective mutual aid planning.
- All acute care hospitals should develop and begin using the HEICS structure.

HEICS commander should be in contact with emergency operation centre coordinating initial care and evacuation.

In India, except for corporate hospitals, the HEICS is not established. It should be mandatory for the hospitals to have HEICS system in place and its details available with emergency operation center and the district disaster plan.

All burn units should have plans to increase bed capacity by 50% in case of disaster and plan to evacuate more stable patients elsewhere in the hospital. If required, trauma facility can be utilized and all surgical beds can also be added to accommodate victims.

Personnel/Staffing

Staffing is the basic requirement for treatment of burns. Advance plan can include short summoning of resident staff. The plan should ensure that there are adequate numbers of trained personnel to give initial resuscitation to burn victim. Education regarding simple management of minor and moderate burns should be imparted to all doctors and nurses out side the burn units so that they become a great resource at the time of disaster. In the hospital, a mixed team of physicians is an asset as they can take care of initial care like establishing intravenous access, intubations, catheterization, initial dressings, etc. The senior surgeon should immediately calculate requirement of staff in coming days, requirement of operation theaters and requirement of dressing change. Volunteers always come whenever there are disasters, Goodness in people is spontaneous. There should be plan to coordinate and supervise these volunteers. A team of hospital staff should be designated to do so, so that requirement by the hospital can be communicated to the volunteers and NGOs. They can be great asset and can serve as clean up crew, help in transport, traffic control, stretcher bearers, managing relatives and friends, give information about victims, managing the dead and help disposal of bodies.

Advanced Burn Life Support (ABLS)

Advanced burn life support (ABLS) developed by American Association of Burns is an excellent tool. It is desired that NABI develops such a course and through its own effort and the burns units all over the country imparts such training. Trained personnel are scarce in disaster; so they should be used wisely. Having single caregiver, usually a nurse or paramedic, taking initial care, intravenous fluids, pain control, tetanus toxoid, airway maintenance, ventilation and documentation is desirable. In case of remote places with non-availability of burn expert, telemedicine facility can be used to give advice and consultation on daily basis. NABI, with Safdarjung Hospital, New Delhi and other burns centers which are adequately staffed, can establish burn telemedicine centers as national resource.

Arturson gave an analysis of San Juanico tragedy in which 7,000 people were injured, 2,000 were hospitalized, 625 had severe burns, 300 died immediately, 250 died in hospitals, later. 6,000 were evacuated and about 7,000 were involved in rescue in first 48 hours. Of these 200 were fire fighters, 1,000 were physician, 1,300 were paramedics, 1,800 were nurses, 2,000 were military personnel, 750 were drivers and helicopter pilots. The plan should have provision for such large mobilization response and coordination.

Handling of relatives and mass media should be relegated to non care giver personnel. Such resource persons should be identified and should be designated beforehand; they should be well versed with press and multi-lingual. Handling of media and giving regular feedback to them about management is very important and it should be integral part of the disaster management. It helps later in secondary mobilization of resources like blood donation and mobilization of volunteers.

Supplies, Equipment and Stockpiles

It is recommended that the disaster plan should take in consideration the supplies and equipment available. In a resource rich country, supplies and equipment can be warehoused but in a country like India where health facilities are always under crunch, it is not possible to make such warehouse or stockpile. The burn disaster may increase the demand on supply and just on time supply system may not be able to keep pace with the demand. The concept of emergency kits for disaster should be an integral part of the plan and it must make provision for at least 50% surge and its maintenance for few days. It should include all necessary supplies for initial care of burn patient and this involves investment of time and money to initiate and then maintain and rotate it.

Other concept is to maintain emergency kits at a regional or central stockpile depot. These can be used to air drop wherever the need of these kits arise including remote areas and where the rescue and emergency burn management team are dispatched or field hospital has been set up. NABI and other burn experts should approve of such kits and get them stockpiled in regional response centers.

Exercising the Plan

Disaster plans usually fail because the dummy runs or mock exercises are not conducted. The dummy runs help in identifying gaps and evolve methods to remedy them. These exercises are essential for role clarity of the stakeholders concerned at the district level and for synergizing coordination of various emergency support functions. A simulation of disaster should be carried out frequently to keep in preparedness. It should involve pre hospital ambulance and evacuation services, hospital based persons, application of HEICS and field teams which are supposed to reach disaster site. The entire system should be tested for seamless operation. The teams should meet frequently for desk top exercises. In India, now through disaster management planning at all levels the mass casualty training is being imparted although their coordination and actual application is yet to be tested.

Medical Response to Mass Burn Casualty

The most common burn mass casualty usually occurs in urban areas or nearby urban areas. It is only rare to have Ural train gas pipe line tragedy in Russia which occurred in remote inaccessible area. Sometimes, the tragedy can strike in a country or areas where there are no facilities to handle mass burn casualties as happened in Bali Night Club bomb explosions. In India, except for Dabwali fire tragedy, most of the fire accidents leading to burn casualties occurred in urban areas where rescue and transport was rapid but not organized. They all followed load and go policy to nearby medical facility without any triage.This leads to choking of emergency services which are not geared to mange such large numbers and the emphasis shifts to severe burn more than 60% TBSA. Lack of awareness about inhalation injury and being unprepared to handle large numbers of inhalation injuries led to high mortality in Uphaar cinema fire in Delhi.

Medical response depends on information reaching the hospital about the incident. It should happen through emergency operation centers. The other information is about the size of disaster and the type and number of casualties expected so that appropriate HEIC can be initiated.

The medical response through trained paramedics or trauma response team on the scene of incident is done in three phases:

1. Primary Triage
2. Ambulance staging and
3. Establishing first aid post for severely burned.

The rescue and extrication should be ideally handled by fire department or other responders trained in entering buildings on fire and how to handle smoke and low visibility.

The guidelines as enumerated by Masellis for immediate care of burn patient are as follows:

Guidelines for Immediate Care of Burn Victim

- Self control
- Self protection
- Qualitative assessment of burn
- Quantitative assessment of burns
- IV fluid therapy
- Analgesic therapy
- Bladder catheterization
- Pressure relieving incision –escharotomy
- Reexamine the patient
- Hospital or burn center transfer
- Whosoever has been exposed to fire and have burns even minor to be examined for Inhalation burns

After the primary triage, patients are shifted to nearby hospitals as there is no time to carry out expert secondary triage. The policy should be on first come first served basis because true priority cannot be actually assigned till the last patient has arrived there. Transfer to other centers, once the capacity of nearby center is full, should be organized through emergency control center.

Griffith has recommended the following triage and care plan. It can be of use in transferring and ambulance staging (Table 39.1).

TRIAGE CRITERION AND CARE PLAN

Except for minor burns all patients, either in field or on reaching hospital, should receive prompt IV fluids. Prognosis depends on the initial fluid replacement. Effective fluid therapy during first 24 hours gives time for organization and secondary triage to determine further transfer.

The Baxter Shriners formula using ringer lactate is the most straight forward and should be followed in all burns mass causalities. Avoid colloids in first 24 hours. The diuresis should be maintained at 0.5 ml to 1 ml/per kg/per hour. Adequate resuscitation is the key to better survival, junior staff nurses and paramedics can set up IV infusion, catheterization, administer analgesics and document injury. In today's time, digital photography is an efficient way of rapid documentation.

Initially burns can be covered with just a clean sheet. Care should be taken to prevent hypothermia, especially in children. Before any patient is dressed, he should be examined

Table 39.1: Triage criterion and care plan

Triage criterion	*Care plan*
Minor burns /non-critical Site < 10%TBSAfor children < 20%TBSA for adult	Outpatient care, tetanus prophylaxis, dress the wound
Minor burns at critical sites, hand face, perineum	Admit, early operation, special wound care, short hospital stay
20-60% TBSA burned	Admit burn unit, IV fluids, careful monitoring requires burn trained personnel
Extensive burns >60% TBSA associated with inhalation injury trauma or medical illness	High mortality, placed in expectant category, pain medication, psychological support
Inhalation injury, minor burns, associated injuries	Administer oxygen, intubate, ventilate, care of associated injuries needs to be in burn center

by burn physician who makes assessment and documentation of burns. Only then, the patient should be dressed. Immediate coverage and dressing is a waste of time of nurses and other junior staff, if not examined in detail by a burn physician. This is the time when secondary triage is conducted and if needed patient transferred to another center.

The protocol for preparing patient for medical evacuation is as follows:

- Cannulation of several routes for IV fluids of which one should be central, followed by chest X-ray to rule out any complication.
- Monitoring of urine output
- Insertion of nasogastric tube
- Sedation, analgesia, cleansing of wound
- Tracheal intubations, if required
- Prevent heat loss.

The only surgical intervention is escharotomy. Early debridement is not done as the burn surgeon will be busy in life saving measures for other victims. It should be carried out only within a burn unit and can be done till 5 days. Although many units do not start prophylactic antibiotics routinely, in case of mass casualty, antibiotics are started to protect the patients from hospital infection. It is also recommended to start prophylaxis against stress ulcers. Psychological support and counseling of the victim and relatives should be given.

DISASTER PLAN IN ACTION

The National Disaster Management Authority published final State Disaster Management Plan document [SDMP] with details of guidelines in July 2007.The SDMP document included various disasters in its list but has not mentioned burn disaster. It is urgently required that NABI should bring this anomaly to the notice of NDMA. There should be planning for mass burn causalities at national level. The burn physician and burn units are important stakeholders in the disaster management. National Disaster Management Guidelines-Medical Preparedness and Mass Casualties Management (NDMG-MPMCM) Plan should recognize and include planning for burn mass casualty and recognize burn units in government and private hospital as national resource.

Any plan for mass burn causality can basically be considered a generic plan for mass trauma casualties or any plan for mass casualty can have sub-plan catering to burn injury.

At district level in case of fire accident leading to burn injury disaster

What will be done?

How it is to be done?

Who will do it?

The logistics management details to be incorporated and documented in respective burn disaster plan.

The moot question is how will the disaster plan be activated and who will trigger its implementation. This will depend on the first information from the site and as the police, fire department and district authorities reach they will provide information to emergency operation center. Under the guidance of team of expert and leaders trained in disaster management, the disaster would be declared and plan shall be implemented with coordination and precision. In India, either it will be done by district collector or specially designated disaster management officer.

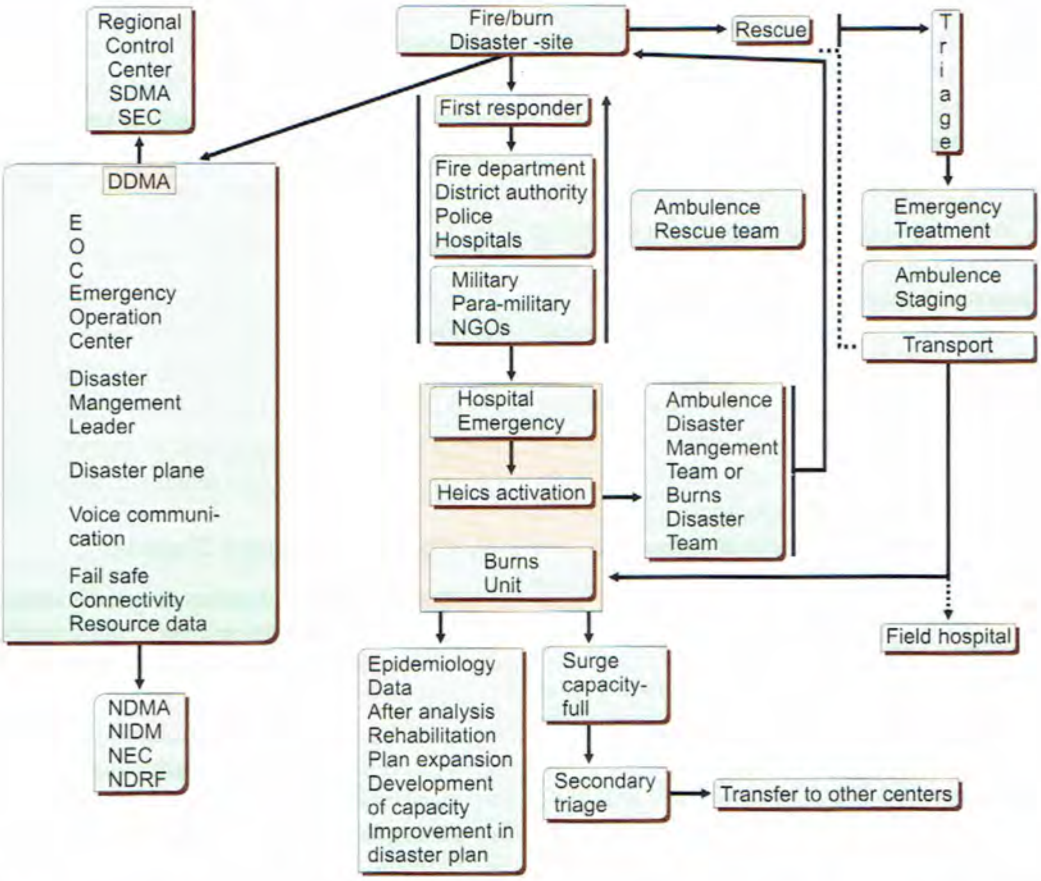
Establishing emergency operation center (EOC) is a must and should be done in every district as recommended in SDMP document. The time bound action plan is must for creating facilities for environment scanning, intelligence gathering, operations and logistics management under one roof for holistic and efficient management of disasters at the state and district level. Arrangements must be made and procedures must be laid down for manning the EOC on 24 × 7 × 365 basis. The plan must also cover in detail the indigenized Incident Command System at the state, district and local body levels. Only states of Maharashtra and Assam have established such center with satellite assisted communication. Without the emergency operation center which is linked to live voice communication to the entire stakeholder in disaster management especially hospitals, ambulance, evacuation services, fire department and police department, it is not possible to respond to any disaster in a coordinated manner.

At emergency operation center, data should be available about hospitals, their capacity and other health facilities. Some of the district medical officers or some medical personnel from the local hospital should be trained to act as field medical officers and act as link and liasoning for emergency management at the site and evacuation to various hospitals.

At hospital level, all hospitals should have emergency incident command system in place, i.e. HEICS and designated commander should be in constant contact of EOC. Once there is information about the incident:

ALERT IS DECLARED—liasoning done with various departments,

Flow chart 39.3: Burns disaster management plan (as suggested by the author)


Regional Control Center SDMA SEC
Fire/burn Disaster -site
Rescue
T r i a g e
First responder
DDMA
E O C Emergency Operation Center
Disaster Mangement Leader
Disaster plane
Voice communi-cation
Fail safe Connectivity Resource data
Fire department District authority Police Hospitals
Military Para-military NGOs
Ambulence Rescue team
Emergency Treatment
Ambulence Staging
Transport
Hospital Emergency
Heics activation
Burns Unit
Ambulence Disaster Mangement Team or Burns Disaster Team
Field hospital
NDMA NIDM NEC NDRF
Epidemiology Data After analysis Rehabilitation Plan expansion Development of capacity Improvement in disaster plan
Surge capacity-full
Secondary triage
Transfer to other centers

STANDBY—information confirmed, hospital concerned consultant put on standby,

EOC contacts hospital incident commander, ambulance and evacuation agencies,

Disaster management team and burn disaster response team readied.

Initial Response

Emergency teams sent to site, coordinate triage, transfer of patient to medical facility, create surge capacity at the hospital, HEICS activation, discharge patients, postpone routine surgeries, summon staff, nurses paramedics, alert pharmacy, blood bank, equipment for emergency care to be ready.

On Going Management

After initial resuscitation, carry out secondary triage; arrange Operation Theater for escharotomy and other emergency operation. Plan for long treatment of the patient requiring coverage. Schematic plan of burn disaster is given (Flow chart 39.3):

After action and review: After the burn mass casualty incident has been managed, a meeting will be arranged of all the participants and the plan should be reviewed. Otherwise also, all plans should be reviewed every two years. All the communication through EOC is transcripted and put on record. It can be evaluated for coordinating response. The benefit of such after action review is to bring practical changes in disaster plan. In the past, after action report of fire in Hilton Hotel, Las Vegas changed safety and fire prevention of high rise buildings forever. This resulted in pressurized fire escapes systems, fire control centers in high rise hotels, hospitals and other public buildings, fire sprinkle system, extensive alarm system. etc. Emphasizing disaster plan makes better disaster prevention.

In India, no analysis of fire disasters is available but from the judicial inquiries it is known that the worst accidents occurred in temporary structures erected without any safety precaution. We still have not been able to impose safety standard for the temporary structures erected.

National Burn Mass Casualty Management System

Training and Community Preparedness Programs: The government of India (GOI) has also taken up an extensive program of training for disaster preparedness and response. The training programs have been designed for the officials of the government at different levels, NGOs, and elected representatives of Local Bodies. The Disaster Management Centers are playing a pivotal role in developing the training literature and organization of training program. In the next two to three years, an intensive training schedule will be drawn up for orienting all the concerned agencies in different areas of disaster preparedness and response. The GOI also proposes to take up community disaster preparedness programs on a pilot basis in different areas of the state. This program will be implemented through the selected NGOs for identified disaster spots in the states and is aimed at building up the mitigation measures into community life.

Developing leadership is the most crucial and important aspect and biggest asset a community can build for tackling mass casualty. This involves knowledge to initiate disaster plan, provide link between medical care system and incident response. A team of persons should be trained to provide disaster-related leadership knowledge and skills and ability to match hospital and system specific resource, knowledge of surge plan, resources and techniques in a particular part of the city or state.

International Applications

In recent years, with rise of international terrorism which is trans borders, it has been noticed that there has been more international cooperation in disaster management. Especially in case of Bali bombings, large patients were shifted to Australia and treated there. In the past also, United States of America, United Kingdom, France, Israel and Cuba have sent their teams for international medical relief. Sending such teams help in tiding away the disaster response need but also bring exchange of new ideas, learning, teaching and sometimes upgrading the host countries medical systems. National Academy of Burns-India along with Government of India should form such burn disaster international relief teams. These teams should be self sufficient, should carry their own bulk supplies and they should not be burden on host country. The required equipment should be stockpiled and kept ready to be retrieved and airlifted with the team on short notice. Such team has to be in state of preparedness

LEGAL FRAME WORK

Although the disaster management has had paradigm shift with enactment of national *Disaster Management Act*, 2005 by Government of India which provides for the creation of a policy, legal and institutional framework, backed by effective statutory and financial support, the details of the following relevant points remain to be worked out by states.

Authority to Declare Disaster

- Induction and triggering disaster management.
- Legal responsibility to treat.
- To admit and treat without questioning financial implications.
- Liability of health department not to be held liable for any criminal and civil suits whosoever participates in disaster management.
- Power to quarantine or restrict movement.
- Compelling provider to participate in disaster management.
- Stream lined financial issues with provider and the victim.

As the states develop their disaster management plans and enact it as law under DM Act 2005, most of these issues will be resolved.

Principles of disaster management: For preparing any burns disaster plan one should follow the principles. The disaster management and the principles are as follows:

- Prevention
- Preparedness
- Disaster profiles
- Disaster patterns
- Planning and preparation for effective multi-disciplinary response
- Mobilization of multi-sectoral manpower resource
- Risk assessment
- Post-emergency phase
- Reconstructive phase
- Community and institutional involvement

CONCLUSION

India is developing its national disaster management.

National disaster management Act, 2005 has been enacted.

It is mandatory for the states to enact their state disaster management plans.

National Disaster Management Authority (NDMA) and National Institute for Disaster Management (NIDM) are the key bodies for disaster management in India.

There is a need to include burn disaster in the plans.

Characteristics of the pathological conditions affecting burn victims, respiratory complications and associated poly trauma in a burn disaster are different from other type of disasters.

The burn disaster should be addressed systematically.

Legislation of disaster management should include burn disaster.

NABI should become a stakeholder in disaster management plans. It has capacity to be the component in national disaster readiness for mass burn incidents.

EOCs at district level are the basic building blocks of disaster management.

All hospitals should develop their HEICS program and implement and rehearse it regularly.

Recommendation for initial management, triage and further treatment in burn units must be developed for our country.

Physician at all levels must be trained in triage and initial management of burns.

Community at large and all stakeholders should be trained in disaster management.

Awareness programs should be conducted regularly for community involvement; every citizen should have ability to deal with emergency.

This article is based on publications of Government of India, viz.,

1. Disaster Management Act, 2005 document,
2. State disaster management plan recommendations of National Disaster Management Authority published by Government of India, July 2007; and
3. Disaster Management plan, State of Maharashtra and text and diagrams from the same have been used.

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Appendices

APPENDIX 1.1: COMPOSITION OF COMMON IV FLUIDS

Crystalloids

Name	*Na^+*	*K^+*	*Cl^-*	*HCO_3^-*	*Misc*	*CHO(g/l)*	*Pr (g/l)*	*MJ/l*	*Osmolal*	*pH*
N. Saline	154	0	154	0	0	0	0	0	308	5.0
Glucose 5 %	0	0	0	0	0	50	0	0.84	280	4.0
Ringer Lactate	131	5	112	29	Mg 1Ca 1 Lactate 28 mM	0	0	0.04	280	6.5
$NaHCO_3$	1000	0	0	1000	0	0	0	0	2000	8.0
Glucose 4% Saline 0.18%	31	0	31	0	0	40	0	–	284	4
Glucose 5% Saline 0.45%	77	0	77	0	0	50	0	–	431	4

Concentration of ions given as mmol/L.

Colloids

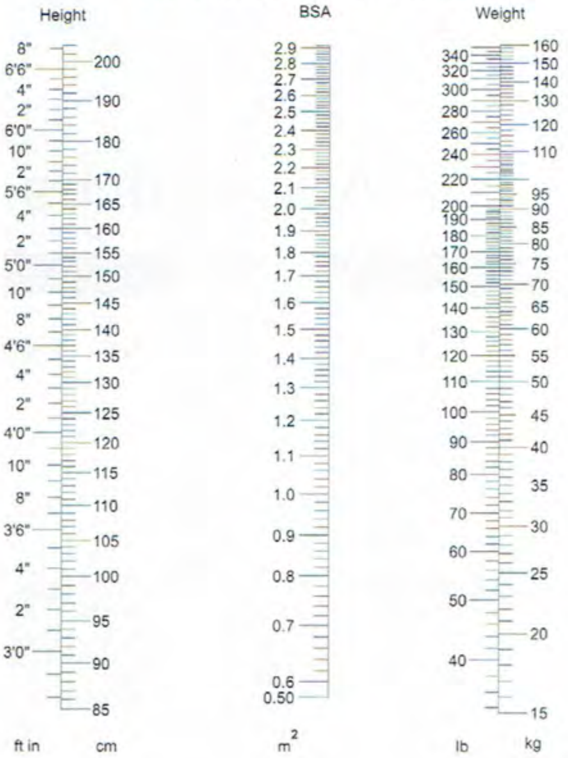
Name	*Na^+*	*K^+*	*Cl^-*	*Misc*	*CHO (g/l)*	*Pr (g/l)*	*MJ/l*	*pH*	*Onc. Pres.*	*$t^{1/2}$*
Human Albumin 4% sol	150	2	120	-	0	19	40	7.4	275	12 hrs
Hemmaccel (succinylated gelatin)	145	5.1	145	Ca 6.3 Gelatin 40 g	0	35	0	7.4	370	5 hrs
HAES	154	0	154	Starch 60 g	0	0	0	5.5	310	17 days

Concentration of ions given as mmol/L.

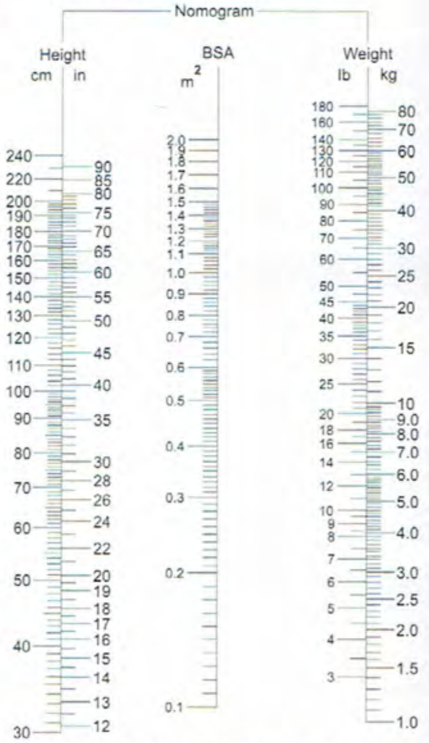
$t_{1/2}$ half life in plasma

APPENDIX 1.2: BODY SURFACE AREA

Dubois nomogram for body surface area



Nomogram for body surface area for children



Approximate areas of the body surface in children at various ages

Age (yr)	Area (m^2)

APPENDIX 1.3: NORMAL BIOCHEMICAL VALUES

Substance	*Traditional units*	*SI units*
Alanine aminotransferase (SGPT)	0-35 U/L	0-0.58 μKat/L
Albumin	4-6 g/dl	40-60 g/L
Alkaline phosphatase	F—30-100 U/L	0.5-1.67 μKat/L
	M—45-115 U/L	0.75-1.92 μKat/L
Amylase	0-130 U/L	0-2.17 μ Kat/L
Aspartate aminotransferase (SGOT)	0-35 U/L	0-0.58 μ Kat/L
Bicarbonate	22-26 mEq/L	22-26 mmol/L
Bilirubin — total	0.1-1.0 mg/dl	2-18 μmol/
— conjugated	<or=2 mg/dl	<or = 4 μmol/L
Blood urea nitrogen(BUN)	8-18 mg/dl	3-6.5 mmol/L
Calcium — total	8.5-10.5 mg/dl	2.2-2.6 mmol/L
— ionized	2.2-2.3 mEq/L	1.10-1.15 mmol/L
Chloride	95-105 mEq/L	95-105 mmol/L
Creatinine	0.6-1.5 mg/dl	0.05-0.13 mmol/L
Glucose — fasting	70-100 mg/dl	3.9-6.1 mmol/L
Magnesium	1.8-3.0 mg/dl	0.8-1.2 mmol/L
Osmolality	280-296 mOsm/kg	280-296 mmol/L
Phosphate	2.5-5 mg/dl	0.80-1.60 mmol/L
Potassium	3.5-5.0 mEq/L	3.5-5.0 mmol/L
Sodium	135-147 mEq/L	135-147 mmol/L
Total protein	6-8 g/dl	60-80 g/L

APPENDIX 1.4: BODY BUILD AND BLOOD VOLUME IN ADULTS

Body build	*Average blood volume (ml/kg)*	
	Males	*Females*
Thin	65	60
Normal	70	65
Muscular	75	70
Obese	60	55

APPENDIX 1.5: FLUID COMPOSITION OF BODY COMPARTMENTS

Estimates of Blood Volume	
Infant	90 ml/kg body weight
Child	80 ml/kg body weight
Adult Male	70 ml/kg body weight
Adult Female	60 ml/kg body weight
Total Water Content	
Adult Male	60 % body weight
Adult Female	55 % body weight
Extracellular Fluid Volume	35 % total water content
Intracellular Fluid Volume	65 % total water content

APPENDIX 1.6: COMPOSITION OF DIFFERENT BODY FLUIDS

Parameter	*Males*	*Females*
Total body water	600 ml/kg	500 ml/kg
Interstitial fluid	160 ml/kg	160 ml/kg
Blood volume	70 ml/kg	65 ml/kg
Erythrocyte volume	33 ml/kg	27 ml/kg
Plasma volume	37 ml/kg	38 ml/kg
Hematocrit	47% (mean) 40-54%	42% (mean) 37-47%

APPENDIX 1.7: NORMAL VALUES FOR COAGULATION TESTS

Partial thromboplastin time	35-45s
Prothrombin time	12-14s
Bleeding time (platelet function)	2-9 min
Plasma fibrinogen	1.5-4.0 g/L
Fibrin degradation products(FDPS)	<10 mg/L

APPENDIX 2.1 AVAILABLE ENTERAL FORMULAE

Presently a wide variety of ready to use commercially prepared diets are available in the market to meet the requirements. However, ready to use feeds are expensive and in our country we still prefer to use blended home made diets.

Example of Home Made Blended Feed

1000 ml milk/buttermilk + 4 eggs + 4 bananas +10 tablespoons of sugar or honey

Final composition of the above is approximately 1700 ml and it provides 1 Kcal/ml of energy, 60 gm of protein and 350 gm of carbohydrate.

Commercially Available Enteral Formulae Include

- Standard formulae (e.g. Fresubin) provide 1kcal /ml.
- Special formulae (Nutrilite) have high essential amino acids content.
- Fiber enriched formulae (Acti-base)
- High lipid content formulae (Pulmocare)
- Glutamine enriched formulae

Table 2.1A: Commercially available protein rich powder with nutritive values

Food product	*Dose*	*Nutritional Value*
Fresubin	50 gm + 180 ml water	E-220 kcal Protein – 8.3 gm Fat – 7.4 gm 1 kcal/ml
Threptin Diskettes	Approx. 5 gm	Protein – 1.5 gm Carbohydrate – Fat – 0.7 gm 22 Calories/Diskette
Nutrilite	10 gm 1 scoop	Protein – 8 gm Carbohydrate – 0.5 gm Fat – 0.3 gm Calories 36 kcal
Acti-base	2 scoops	Protein – 16.5 gm Carbohydrate 15 gms Fat 0.7 gm Calories – 132 kcal
GRD	100 gm/2 tsf =30 gm	Protein – 18.5 gm Carbohydrate – 63 gm Calories – 425 kcal/ 100 gm

Table 2.1B: Commercially available amino acids and lipids

Trade name	*Dosage*	*Protein*	*Non-protein calories*
Alamine –N	200 ml over 2-3 hr	6 gm	24 kcal
Alamine –SE (in patients with oliguric renal failure)	20 ml	1.64 gm	6.5 kcal
Alamine –SN	20 to 500 ml	2 gm	80 kcal
Amino Drip	200 to 500 ml	7.5 gm/100 ml	30 kcal /100 ml
Hermin	200 to 800 ml	100% essential amino acids	500 ml 10% = 450 kcal
Lipofundin (5 to10 %)	100 ml to 500 ml 10 to 20 ml /kg/day start with 15 to 20 drops /min. after 15 minutes increase to 45 drops/min.		20% = 900 kcal

APPENDIX 2.2: NUTRITIVE VALUE OF INDIAN FOODS

	Average portion	*weight*	*protein*	*Fat*	*Carbohydrate*	*Calories*
Beverages						
Coffee: Cows milk ,sugar 2 tsf	1 Cup	200	1.1	1.1	11.4	60
Lemonade: Fresh lime juice 2 tbl. Sp., sugar 3 teaspoon	1 Glass	240	-	-	17.4	73
Tea : Cow's milk 2 tablespoons sugar 2 teaspoons	1 Cup	200	0.9	1.1	11.4	60
Cereals and cereal food						
Cornflakes	1 Cup	25	2.1	0.1	2.1	95
Jowar (sorgum vulgare cholam)	1 small Chapatti	45	3.1	0.6	22.2	106
Maize 30 gm flour	1 Chapatti	45	3.3	1.1	19.8	102
Ragi 30 gm flour	1 Chapatti	45	2.1	0.4	21.6	98
Rice, milled, boiled (3 tbl. spn .dry)	1 Cup	-	2.9	0.1	30.2	138
Wheat Bread	1 Slice	30	2.3	0.5	15	75
Wheat 'chapati' (15 gm flour)	1 thin	-	1.2	0.1	8	40
Wheat 'Parantha' 60 gm and fat 2 teaspoons	1	-	4.8	10.4	36	256
Milk and Milk Products						
Buttermilk, Buffalo's (curd 1 cup making 3 cups of butter milk)	1 Cup	200	2.7	4.6	2.1	62
Cheese, cheddar	1Piece	28	7	9	0.6	111
Cheese cottage	1 round tablespoon	28	5.5	0.1	0.6	27
Cream light	1 tablespoon	30	0.8	5.4	1.2	56
Curd, buffalo milk	1Cup	200	8	14	6.3	182
Ice cream	1 helping	100	4.1	11.3	19.8	196
Milk Buffalo's	1Cup	200	8	14	6.3	182
Milk Buffalo's skimmed	1Cup	200	8.4	0.2	10.8	78
Milk Cow's	1Cup	200	7	9	9.6	160
Milk Cow's skimmed	1Cup	200	7.2	0.2	10	70
Milk condensed sweetened	1 tablespoon	20	1.5	1.7	10.1	62
Milk Goat's	half cup	100	3.3	4.1	4.7	75
Milk whole Powdered	1 tablespoon	7	1.9	1.9	2.7	35
PULSES, DRIED PEAS AND BEANS						
Bengal Gram (urad dal)	1Cup cooked thin dal		200	7	2.3	14 105
Black Gram (urad dal)	1Cup cooked thin dal		200	7	2.3	14 105
Green Gram (murg dal)	1Cup cooked	200	7	2.3	14	105
Field bean (val dal)	half Cup	100	6	0.4	17.3	92
Lentil (masur dal)		100	22.9	11.4	58.1	340
Peas Dried						
Red Gram (tur dal)						
Beans baked						
Peas Roasted						

APPENDIX 2.3: DIET CHART FOR BURN PATIENTS AND THEIR NUTRITIVE VALUE (TABLE 2.3A)

Calculations: 3,000 to 4,000 kcal + 180 gm of Protein can be given by oral route. According to Currerie Formula:

For a 40% Burns

50 kg weight}- Calories required = 20 × body weight + 70 × % of burns

Calories = 20 × 50 + 70 × 40 = 3800 kcal

Protein = 1 gm/kg + 3 gm/% of burn

Protein = 50 + 3 × 40 = 170 gm

Table 2.3A: Diet chart

	Main food Group	*Weight and Measures*	*Calories*	*Protein*
Pre-breakfast	Tea with sugar	125 ml	40	
	+			
	4 Marie biscuits	4	50	
Breakfast	Milk + 2 tsf sugar +2 tsf Proteinex	200 ml + 30 gm + 30 gm	200 + 120	10 + 3 gm
	Porridge/Cornflakes Dalia	2 Katori	410	2 gm
	Stuffed Parantha	90 gm + 40 gm	210 + 100	20 gm + 2 gm
	Bread 2 slices + 2 egg	40 gm	100	10 gm
Pre-lunch	Sprout or Roasted Chana	1 Katori	140	6 gm
	Cheese	2 Cubes	100	10 gm
	Milk + 2 tsf Proteinex+ 2 tsf Sugar	200 ml	320	13 gm
Lunch	Cereal	Chappati 4 medium size 120 gm +2 tsf Ghee(30 gm)	600	
	Leafy Vegetables	2 Cups	40	
	Dal or whole Pulses/Non Veg.	3/4 Katori cooked	100	6 gm
	Curd/ Raita /Butter Milk	1/2 Cup	40	
	Fruit	Banana (big)	80	
Evening Snacks	Milk + 2 tsf Proteinex + 2 tsf Sugar	200 ml + 4-5 Marie Biscuits	320	13 gm
	Cereal	Dhokla /Veg. Sandwitch/ Sprouts/Roasted chana	140	
Predinner	Chicken soup	1 Cup 200 ml	185	10 gm
	Corn soup (sweet)	1 Cup	120	
	Dal soup	1 Cup	169	6 gm
Dinner	Cereal	Chappati (4 med. Size)	600	
	Leafy Vegetable	1 Katori (30 ml)	50	
	Dal or Pulses	1 Katori (cooked)	100	6 gm
	Curd	½ Cup	40	
Desserts				
Rasgulla or	1 (150 gm)	140		
Gulab Jamun or	25 gm	200		
Laddo or	30 gm	160		
Mango Juice	200 ml (Big Glass)	150		

Use 4 Tsf Ghee Per Day

Total Calories = 4,000 Kcal; Total Protein = 117 gm

APPENDIX 2.4: TOTAL PARENTERAL NUTRITION

TPN can be given as separate components like amino drip, *intra lipid*, 10% dextrose or as premixed multi-component solutions *like Clinomel*, which are now available and have reduced the potential for error and metabolic aberration.

Maximum rate of dextrose administration should be 4-6 mg/kg/min to avoid the risk of hyperglycemia.

If carbohydrate is given as the only source of energy, it can lead to:

- Deficiency of essential fatty acids
- Deficiency of fat soluble vitamins
- Excess CO_2 load
- Hepatic steatosis
- Increases osmolarity of solution

Instruction charts should be maintained stating the following details of parenteral nutrition.

- Total energy calculated on body weight basis.
- Substrate distribution (volume of carbohydrate, fat and protein)
- Fluid volume.
- Rate of delivery.
- Hang time.
- Total time frame.
- Steps to be taken in case of accidental change in rate of delivery.

It is said that to prevent the complications of parenteral feeding, like over feeding of intravenous nutrients which may lead to hepatic disorders, fluid retention and metabolic derangements, the required volume for the parenteral nutrition is determined by the rate of substrate utilization as opposed to a predetermined energy goal, as demand of energy may reach to 50 kcal/kg/day. It is important to keep a rational balance and not overload the patient with huge amounts of energy.

The maximum oxidation of glucose in liver is when given at a rate of 4-6 mg/kg/min. If given beyond this rate, it is not assimilated but deposited as fat beneath the skin.

Proteins at 2.5-4 gm/kg can be given, increasing gradually to the desired goal.

Fat at 20% intralipid is given but not during acute phase of shock. Starting with 0.5 gm/kg/hour, the maximum rate of 0.119gm/kg/hour or 1.0 to 1.5 gm/kg/day should not be exceeded.

Intralipid administration should not exceed more than 3.6 gm/kg/day. Calculate **daily volume requirement** on the basis of;

- All on-going losses, including insensible loss
- Input-output chart
- Alternatively, 30 to 40 ml/kg body weight
- Caution in patients with renal dysfunction and congestive heart failure

  For infants and children < 20 kg =1.7 ml/kg/hour

  For children> 20 kg =1.5 ml/kg/hour.

This is started at 60 ml/hr and increased to 80 to 100 ml/hr after 4 to 6 hours. PN should not be started out of hours (evenings and weekends).Lipid emulsions when given alone should be completely infused within 12 hours of hanging. Lipids when administered as total nutrient admixture can be hung for 24 hours.

If parenteral nutrition is interrupted due to any reason, clear instructions should be stated to administer 10% dextrose solution at the same rate to prevent sudden hypoglycemia caused by high endogenous insulin. Complications of intravenous hyperalimentation are greater than with enteral route.

APPENDIX 2.5: STANDARD PARENTERAL SOLUTION

Parenteral nutrition may be delivered either through all in one system like *OliClinomel* which is a preferred way of administering parenteral nutrition in our unit where all macronutrients and micronutrients required for 24 hours are infused from one bag or through multiple bottles. There are fewer chances of infection and metabolic aberrations in using single bag system.

Oliclinomel

Oliclinomel N4-550E, emulsion for infusion.

Qualitative and Quantitative Composition is given in Tables 2.5A and 2.5B.

This medicinal product is presented in the form of 3 compartment bag.

It is available in 4 different presentations and volumes.

Table 2.5A: Qualitative and quantitative composition of oliclinomel

Compartment	1000 ml	1500 ml	2000 ml	2500 ml
Lipid emulsion	200 ml	300 ml	400 ml	500 ml
Amino acid solution	400 ml	600 ml	800 ml	1000 ml
Glucose solution	400 ml	600 ml	800 ml	1000 ml

Table 2.5B: After the contents of the three compartments have been mixed, the final mixture for each of the bag

Per bag	*1 Liters*	*1.5 Liters*	*2 Liters*	*2.5 Liters*
Nitrogen (g)	3.6	5.4	7.3	9.1
Amino acids (g)	22	33	44	55
Total calories (kcal)	610	910	1215	1520
Non-protein calories (kcal)	520	780	1040	1300
Glucose calories (kcal)	320	480	640	800
Lipid calories (kcal)	200	300	400	500
Non-protein calories/ nitrogen Ratio (kcal /gm N)	144	144	144	144
Sodium (mmol)	21	32	42	53
Potassium (mmol)	16	24	32	40
Magnesium (mmol)	2.2	3.3	4.4	5.5
Calcium (mmol)	2	3	4	5
Phosphate (mmol)	8.5	13	17	21
Acetate (mmol)	30	46	61	76
Chloride (mmol)	33	50	66	83
pH	6	6	6	6
Osmolarity (mmol/L)	750	750	750	750

The final mixture allows parenteral nutrition for adults and children above 2 years of age when oral or enteral nutrition is not possible, insufficient or contraindicated.

Dosage depends on metabolic requirements, energy expenditure and the patient's clinical condition.

Maximum Daily Dose in Adults

The maximum daily dose is 40 ml/kg body weight (equivalent to 0.88 g of amino acids, 3.2 g of glucose and 0.8 g of lipids per kg) i.e. 2800 ml of the emulsion for a patient weighing 70 kg.

Maximum Daily Dose in Children Above 2 Years

The maximum daily dose is 100 ml/kg body weight (equivalent to 2.2 g of amino acids, 8 g of glucose and 2 g of lipids per kg body weight)

As a general rule, do not exceed doses of 3 g/kg/day of amino acids and/ or 17g/kg/day of glucose and/or 3g/kg/day of lipids, except in spacial cases. The dosage is based on fluid intake and daily nitrogen requirements.

Special Warnings

Water and electrolyte equilibration and metabolic disorders must be corrected before starting the infusion. This product does not contain vitamins or trace elements. They should be supplemented

APPENDIX 2.6: NUTRITIONAL ASSESSMENT CHART (TABLE 2.6A)

Stepwise Calculation of Nutritional Requirement

Surgeons and residents involved in management of burns should learn to prescribe a nutritional regimen in a methodic and scientific manner as follows:

1. Before starting nutritional support, patients with shock should be adequately resuscitated and gross fluid and electrolyte imbalance corrected. Mild degree of fluid and electrolyte imbalance can be corrected with ongoing nutritional support.
2. Decide on the route of administration for nutrition, plan the oral/enteral/or parenteral nutrition or partial parenteral nutrition.
3. Calculate the energy requirements of burn patients according to currerie formula depending on the size of burn wound and age of the patient
4. These formula or equation for calorie estimation often overestimates the calorie requirements.
5. To avoid this and simplify it, one can easily calculate the calorie protein needs according to appropriate stress level taking the severity of disease into consideration.
6. Calories are derived from three major substrates—Carbohydrates, fats, and proteins. By and large 50% of calories are derived from carbohydrates, 30% from fats and 20% from proteins. In critically ill patients, certain adjustments are required as mentioned earlier.
7. After finalizing the total calorie and substrate requirements, the total permissible liquid intake has to be calculated. Normal recommendation is 30-40 ml/kg/day. However, liquid intake has to be adjusted in specific conditions such as renal and cardiac failure, where volume load needs to be restricted. Consequently; total calories will have to be given in restricted volume of fluid. In patients on total parenteral nutrition, this becomes a challenge and one will have to resort to using more lipid emulsions as they give higher calories on weight to weight basis.
8. Let us now practically work out a stepwise prescription, taking an actual case scenario, a male patient aged 40 years, weighing 50 kg with 20% burns.

Table 2.6A: Nutritional assessment chart

Name of Patient Age/Sex

Height Weight Ideal Weight

Date of Burn.

Date of Arrival

Total Body Surface Burn. % Full thickness

Estimated Energy Requirement Kcal/Day

Estimated Protein Requirement Gms/Day

Kcal															
Oral															
Enteral															
Parenteral															
Protein															
Oral															
Enteral															
Parenteral															
Na^+/K^+															
Glucose															
BUN															

Step 1

Energy = (25 × wt in kg) + (20 × % burns) E = (25 × 50) + (20 × 20)

E = 1650 kcal

Step 2

Protein requirement in 24 hours

2 gm/kg body wt.

Protein = 2 × 50 = 100 gm

Protein calories = 100 × 4 =400 kcal

So non-protein Energy (NPE) = 1650 – 400 = 1250 kcal

Energy values Protein = 4 kcal /gm

Carbohydrate = 4 kcal/gm

Fat = 9 kcal/gm

Step 3

For all age groups give 40 to 50% of non-protein calories as fat, 50 to 60% as carbohydrate.

50% of 1250 = 625 kcal

Fat required = 625/9 = 70 gm

Carbohydrate required = 625 /4 = 156 gm

Step 4

Daily requirement in 50 kg wt. patient with 20% burn is:

Protein = 100 gm

Carbohydrate = 156 gm

Fat = 70 gm

1 liter of 5% dextrose = 50 gm of monohydrate glucose = 200 kcal

6.25 gm of protein = 1 gm of body nitrogen

Step 5

Calculate fluid Intake in 24 hours

Average requirement in an adult male is 40 ml /kg/day

i.e. 40 × 50 = 2000 ml/day

Step 6

Now prepare standard nutrition (Parenteral/oral/enteral) for 2000 ml

If more than 180 gm of carbohydrate is given/day frequent monitoring of blood glucose is required and insulin may be necessary.

100 gm protein = 1000 ml of 10% amino acids

150 gm of carbohydrate = 750 ml of 20% dextrose

70 gm of lipids = 350 ml of 20% lipofundin

Total volume = 2100 ml giving 1650 calories

The above composition can be conveniently delivered as an All-in-one solution which is commercially available as Clinomel or as multiple bottles available as amino drip, lipofundin and dextrose

Step 7

If the above patient develops renal failure and his protein intake has to be decreased to 40 gm of proteins and 1000 ml of fluid intake /day

40 gm of protein = 400 ml of 10 % aminodrip

150 gm carbohydrate = 300 ml of 50 % dextrose

70 gm lipids = 250 ml of 30% lipids

Final volume = 950 ml giving 1470 calories

It can be given as 50% glucose solution, 30% lipid solution and 10% amino acid solution

Diabetics or patients with glucose intolerance require close monitoring of blood glucose levels and need correction. For them, reduce glucose as energy substrate and instead use lipid regimens.

Insulin may be given according to revised sliding scale by Dickerson (Table 2.6B).

Table 2.6B: Sliding scale for insulin dose management

Finger stick glucose Concentration (mg/dl)	*Amount of regular Human insulin (units)*
Less than 70	0
71 to 125	0
126 to 150	2
151 to 175	4
176 to 200	6
201 to 225	8
226 to 250	10
251 to 275	12
276 to 300	14
more than 300	16

NUTRITIONAL VALUES

NUTRITION READY RECKONER

These are average values for standard recipes found in most books

	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
BEVERAGES														
Hot Tea	34	0.6	1.0	5.7	0.0	31.0	0.0	0.0	7.00	0.01	0.01	0.0	0	1 Tea Cup
Instant Coffee	149	1.0	13.3	6.3	0.0	31.0	0.0	0.0	7.00	0.01	0.01	0.0	0	1 Tea Cup
Cold Coffee (with Cream)	279	3.9	17.0	27.7	0.0	144.0	0.3	487.0	183.00	0.06	0.23	0.1	2	1 Tall Glass
Banana Milk Shake	228	6.2	7.5	33.8	0.0	223.0	0.5	40.0	101.00	0.11	0.37	0.4	7	1 Tall Glass
Mango Milk Shake	237	6.2	7.7	35.6	0.8	227.0	1.3	2067.0	608.00	0.15	0.41	9.0	16	1 Tall Glass
Lemonade	107	0.3	0.3	25.7	0.5	21.0	0.1	0.0	0.00	0.01	0.00	0.0	12	1 Glass
BREAKFAST CEREALS														
Cracked Wheat Porridge	292	10.0	10.4	39.7	2.5	296.0	1.5	152.0	39.00	1.29	0.49	1.3	5	1 Bowl
Semonlina Porridge	293	9.8	10.2	40.4	0.0	291.0	0.8	139.0	36.00	1.24	0.47	0.6	5	1 Bowl
Vermicelli Porridge	294	9.4	10.2	41.1	2.5	292.0	0.9	139.0	36.00	1.24	0.47	0.6	5	1 Bowl
Oat Meal Porridge	217	6.6	6.6	32.8	2.0	154.0	1.0	70.0	18.00	0.80	0.26	0.3	2	1 Bowl
Cornflakes with Milk	291	9.8	10.8	38.7	1.2	290.0	0.9	157.0	41.00	1.28	0.48	0.6	5	1 Bowl
Semolina Upma	147	2.3	7.7	17.1	9.9	11.0	0.5	1.0	0.00	0.04	0.01	0.4	2	1 Bowl
Vermicilli Upma	296	4.0	15.2	35.7	5.4	24.0	1.2	2.0	1.00	0.10	0.03	0.9	5	1 Bowl
Vegetable Upma	192	4.4	9.7	21.7	1.5	22.0	0.9	107.0	27.00	0.20	0.02	1.6	4	1 Bowl
Poha	158	2.8	7.3	20.2	1.5	10.0	4.2	6.0	1.00	0.10	0.02	2.0	3	1 Bowl
Vegetable with Poha	202	4.9	9.3	24.7	2.1	21.0	4.6	111.0	28.00	0.18	0.03	3.1	5	1 Bowl
EGGS														
Boiled Egg	87	6.7	6.7	0.0	0.0	25.0	0.7	300.0	180.00	0.05	0.20	0.1	0	1 Egg
Poached Egg	87	6.7	6.7	0.0	0.0	25.0	0.7	300.0	180.00	0.05	0.20	0.1	0	1 Egg
Fried Egg	160	6.7	14.8	0.0	0.0	25.0	0.7	620.0	260.00	0.05	0.20	0.1	0	1 Egg
Scrambled Egg	172	6.7	15.8	0.8	0.0	57.0	0.7	620.0	267.00	0.06	0.22	0.1	0	1 Egg
Baked Egg	124	6.7	10.8	0.0	0.0	25.0	0.7	460.0	220.0	0.05	0.20	0.1	0	1 Egg
Fluffy Omelette	160	6.7	14.8	0.0	0.0	25.0	0.7	620.0	260.00	0.05	0.20	0.1	0	1 Egg
Cheese and Mushroom Omelette	308	12.9	27.1	3.0	0.0	182.0	1.3	780.0	373.00	0.09	0.42	1.5	1	1 Egg
SOUPS														
Minestrone Soup	90	1.4	5.2	9.4	1.2	43.0	0.7	491.0	123.00	0.07	0.03	0.5	17	1 Bowl
Chicken Sweet Corn Soup	322	25.5	13.5	24.6	6.0	23.0	2.6	10.7.0	93.00	0.21	0.34	8.6	6	1 Bowl
French Onion Soup	208	4.8	11.6	21.1	2.4	102.0	0.8	321.0	110.00	0.08	0.05	0.6	8	1 Bowl
Tomato Soup	82	2.1	4.5	8.3	2.3	101.0	1.3	862.0	216.00	0.25	0.21	0.8	55	1 Bowl
Green Pea Soup	186	9.0	6.4	23.1	2.2	70.0	1.9	375.0	109.00	0.28	0.08	1.1	11	1 Bowl
Spinach Soup	561	3.9	8.9	116.2	5.9	81.0	1.5	5902.0	1475.00	0.06	0.27	0.08	29	1 Bowl

Contd.

Contd.

	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
Mixed Vegetable Soup	146	3.3	9.2	12.5	1.4	124.0	0.9	779.0	225.0	0.11	0.16	0.6	23	1 Bowl
Cream with Tomato Soup	245	5.4	16.6	18.5	2.3	180.0	1.5	984.0	287.00	0.32	0.25	1.0	45	1 Bowl
Cream with Spinach Soup	307	8.5	23.2	16.0	5.1	200.0	2.2	6214.0	1644.0	0.20	0.52	0.9	32	1 Bowl
Cream with Carrot Soup	250	4.6	16.2	21.5	2.0	172.0	1.4	1935.0	531.00	0.17	0.17	0.9	6	1 Bowl
Cream of Pea Soup	342	12.4	18.2	32.2	2.6	132.0	2.4	590.0	188.00	0.44	0.17	1.4	15	1 Bowl
Cream with Potato Soup	282	5.4	13.1	35.7	3.1	118.0	1.1	415.0	144.00	0.24	0.17	1.6	21	1 Bowl
Cream with Mixed Vegetable Soup	263	7.5	13.4	28.0	3.2	179.0	2.0	1160.0	331.00	0.33	0.24	1.5	45	1 Bowl
Cream with Mushroom Soup	308	6.6	22.4	19.9	0.7	136.0	1.5	554.0	189.00	0.13	0.41	2.9	6	1 Bowl
Hot and Sour Soup	181	11.2	9.3	13.2	1.6	65.0	2.8	86.0	22.00	0.22	0.25	23	6	1 Bowl
CEREALS														
Chapati	273	9.7	1.4	55.5	5.6	38.0	3.9	23.0	5.00	0.39	0.14	3.40	0	3 or 4
Plain Parantha	363	9.7	11.4	55.5	5.6	38.0	3.9	23.0	6.00	0.39	0.14	3.40	0	3 Medium
Potato Stuffed Parantha	632	11.2	31.5	75.8	7.9	56.0	4.4	42.0	10.00	0.49	0.15	4.50	16	3 Medium
Pea Parantha	693	20.7	31.5	75.8	7.9	78.0	6.3	148.0	37.00	0.78	0.15	4.70	16	3 Medium
Cauliflower Parantha	577	12.0	31.7	60.9	7.1	74.0	5.0	47.0	12.00	0.44	0.22	4.30	47	3 Medium
Radish Parantha	574	10.8	31.5	61.8	8.2	90.0	4.5	27.0	7.00	0.48	0.16	4.10	20	3 Medium
Daal Parantha	665	16.2	33.1	75.6	9.0	65.0	5.6	62.0	15.00	0.55	0.19	4.20	3	3 Medium
Sprouted Dal Parantha	727	22.1	32.0	87.6	10.2	85.0	6.0	48.0	12.00	0.64	0.24	4.70	2	3 Medium
Paneer Parantha	713	20.9	43.9	58.5	5.9	173.0	4.0	243.0	61.00	0.45	0.15	3.50	4	3 Medium
Onion-Green Chilli Parantha	594	10.9	31.5	66.6	7.1	85.0	4.5	23.0	6.00	0.57	0.15	3.80	11	3 Medium
Keema Parantha	712	24.8	42.1	58.4	5.9	169.0	6.2	25.0	13.00	0.55	0.25	9.00	3	3 Medium
Methi Parantha	652	14.2	32.4	75.8	8.1	293.0	7.2	1230.0	303.00	0.52	0.32	4.50	0	3 Medium
Pooris (10 g oil absorbed)	375	9.2	12.1	57.4	4.0	28.0	3.0	42.0	11.00	0.24	0.10	2.70	0	3 Medium
Daal Stuffed Pooris	427	12.8	12.3	66.3	51.5	5.0	3.6	47.0	12.00	0.31	0.13	3.00	0	4 or 5
Palak Pooris	388	10.2	12.4	58.8	64.9	6.0	3.6	2832.0	708.00	0.26	0.23	2.9	14	4 or 5
Besan Pooris	380	11.2	13.0	54.6	5.5	35.0	3.6	62.0	16.00	0.32	0.12	2.7	0	4 or 5
Pea Pooris	431	13.6	12.1	66.9	5.0	40.0	3.9	91.0	23.00	0.39	0.10	3.2	5	4 or 5
Pea Kachori (10 g oil absorbed)	215	6.9	7.4	30.2	1.8	17.0	1.6	49.0	13.00	0.16	0.03	1.1	5	2
Khasta Kachori (10 g oil absorbed)	441	11.4	18.9	56.3	3.2	45.0	2.4	163.0	41.00	0.16	0.8	1.8	0	2
Naan	200	6.7	2.1	38.5	1.5	60.0	1.4	31.0	9.00	0.19	0.10	1.2	1	1
Bhatura	176	5.6	3.8	29.9	1.2	28.0	1.2	51.0	27.00	0.06	0.07	0.1	0	2 or 3
Boiled Rice	277	6.0	0.8	61.4	3.6	8.00	2.6	2.0	0.40	0.17	0.13	3.1	0	1 Full plate

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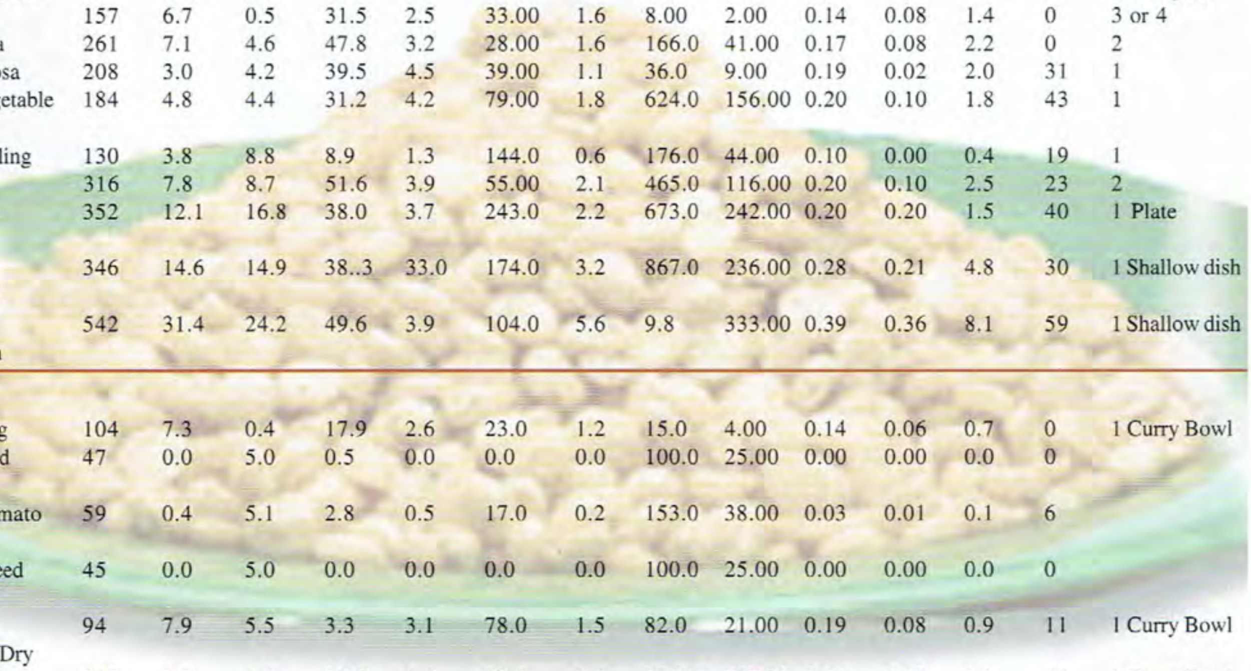
	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
Curd Rice	484	15.1	12.5	77.8	5.3	253.0	3.8	66.0	26.00	0.33	0.41	3.7	2	1 Full plate
Zeera Pulao	400	8.2	10.5	68.1	4.4	25.00	2.9	78.0	45.00	0.22	0.18	3.5	6	1 Full plate
Mixed Vegetable Pulao	523	17.6	9.0	92.9	7.2	71.0	5.2	126.0	32.00	0.60	0.15	4.6	21	1 Full plate
Pea Pulao	510	12.1	9.1	94.8	8.1	108.0	4.6	1033.0	258.00	0.44	0.17	4.8	28	1 Full plate
Channa Daal & Vegetable Pulao	529	15.1	10.1	94.3	8.5	102.0	5.3	1014.0	254.00	0.47	0.19	4.6	14	1 Full plate
Black Channa Pulao	464	10.9	10.2	82.1	7.5	82.00	4.0	49.0	11.00	0.28	0.17	4.1	6	1 Full plate
Mushroom Pulao	411	9.7	9.7	71.2	4.3	38.00	4.4	2.0	0.40	0.31	0.55	8.1	9	1 Full plate
Paneer Pulao	520	13.3	20.4	70.9	4.3	272.0	4.9	2.0	0.40	0.21	0.13	3.3	6	1 Full plate
Mutton Pulao	622	22.6	28.5	68.7	4.3	216.0	5.1	13.0	12.00	0.37	0.29	8.8	6	1 Full plate
Chicken Pulao	661	27.9	30.5	68.7	4.3	110.0	4.6	88.0	90.00	0.33	0.34	11.4	6	1 Full plate
Idli	157	6.7	0.5	31.5	2.5	33.00	1.6	8.00	2.00	0.14	0.08	1.4	0	3 or 4
Plain Dosa	261	7.1	4.6	47.8	3.2	28.00	1.6	166.0	41.00	0.17	0.08	2.2	0	2
Masala Dosa	208	3.0	4.2	39.5	4.5	39.00	1.1	36.0	9.00	0.19	0.02	2.0	31	1
Mixed Vegetable Dosa	184	4.8	4.4	31.2	4.2	79.00	1.8	624.0	156.00	0.20	0.10	1.8	43	1
Paneer Filling	130	3.8	8.8	8.9	1.3	144.0	0.6	176.0	44.00	0.10	0.00	0.4	19	1
Uttapam	316	7.8	8.7	51.6	3.9	55.00	2.1	465.0	116.00	0.20	0.10	2.5	23	2
Beans and Macaroni	352	12.1	16.8	38.0	3.7	243.0	2.2	673.0	242.00	0.20	0.20	1.5	40	1 Plate
Spaghetti Bolognese	346	14.6	14.9	38..3	33.0	174.0	3.2	867.0	236.00	0.28	0.21	4.8	30	1 Shallow dish
Chicken Chowmein	542	31.4	24.2	49.6	3.9	104.0	5.6	9.8	333.00	0.39	0.36	8.1	59	1 Shallow dish
PULSES														
Dal Moong	104	7.3	0.4	17.9	2.6	23.0	1.2	15.0	4.00	0.14	0.06	0.7	0	1 Curry Bowl
Cumin Seed Baghar	47	0.0	5.0	0.5	0.0	0.0	0.0	100.0	25.00	0.00	0.00	0.0	0	
Onion Tomato Baghar	59	0.4	5.1	2.8	0.5	17.0	0.2	153.0	38.00	0.03	0.01	0.1	6	
Mustard Seed Baghar	45	0.0	5.0	0.0	0.0	0.0	0.0	100.0	25.00	0.00	0.00	0.0	0	
Urad Sp. Dehusked Dry	94	7.9	5.5	3.3	3.1	78.0	1.5	82.0	21.00	0.19	0.08	0.9	11	1 Curry Bowl
Arhar Daal with Spinach	159	7.7	5.9	18.7	4.4	58.0	1.4	2830.0	707.00	0.50	0.19	1.1	14	1 Curry Bowl
Channa Daal with Ghia	117	6.3	1.7	19.1	3.4	27.0	1.8	39.0	10.00	0.16	0.06	0.8	0	1 Curry Bowl
Sambar	119	5.6	2.5	18.4	3.2	45.0	1.7	88.0	22.00	0.13	0.08	0.9	19	1 Curry Bowl
Green Gram Whole	100	7.2	0.4	17.0	1.2	37.0	1.3	28.0	7.00	0.14	0.08	0.6	0	1 Bowl
Dry Khatta Channa	232	6.0	11.7	25.6	4.8	83.0	1.9	369.0	91.00	0.16	0.07	1.3	14	1 Bowl
Rajma Curry	268	10.5	11.0	31.8	3.7	162.0	2.6	193.0	49.00	0.29	0.12	1.2	20	1 Bowl
Lobia Curry	259	10.9	10.9	29.3	2.8	89.0	4.1	182.0	46.00	0.31	0.12	0.9	19	1 Bowl
Besan Gatte Curry	497	21.3	30.6	34.0	5.5	255.0	2.9	367.0	96.00	0.34	0.22	1.4	14	1 Bowl
Besan Kadhi with Pakoris	408	9.9	30.2	24.1	3.7	140.0	2.2	73.0	23.00	0.21	0.19	1.0	1	1 Bowl



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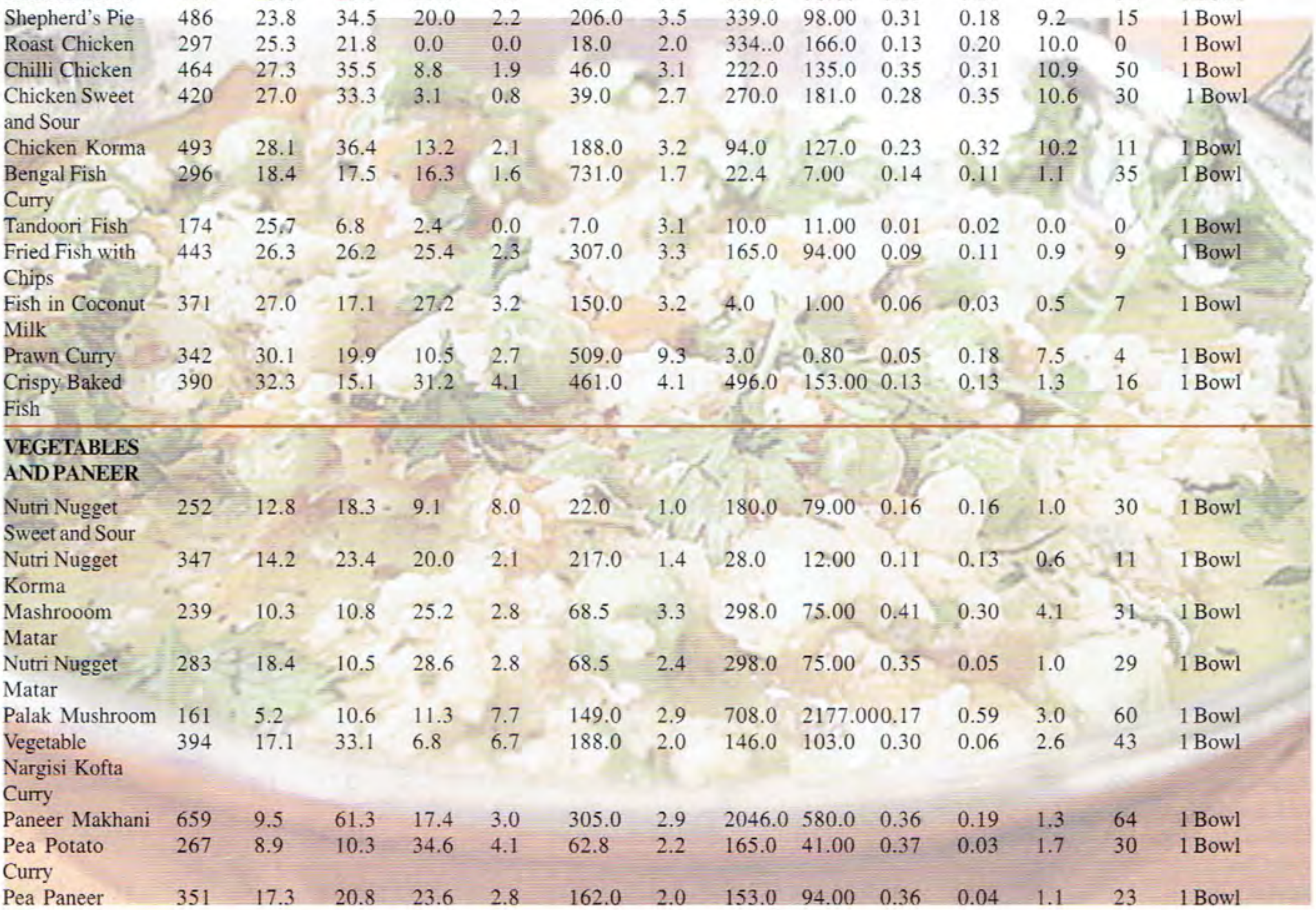

	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
MEATS														
Keema Matar	411	26.9	23.7	22.6	2.8	215.0	4.9	298.0	84.00	0.53	0.19	8.0	29	1 Bowl
Roganjosh	404	25.0	30.6	7.2	0.8	276.0	4.0	114.0	45.00	0.31	0.27	8.0	12	1 Bowl
Mutton Korma	439	24.0	34.7	7.7	0.8	259.0	3.7	149.0	54	0.30	0.22	8.50	6.0	1 Bowl
Palak Meat	374	19.1	28.4	10.6	5.2	290.0	3.8	5600.0	1410	0.24	0.46	620	34.0	1 Bowl
Shahi Keema Kofta Curry	508	23.8	41.3	10.2	1.5	238.0	4.3	217.0	65	0.34	0.21	8.70	23.0	1 Bowl
Boti Kabab	365	23.6	27.8	5.0	0.5	232.0	3.6	8.0	19	0.24	0.22	8.30	9.0	1 Bowl
Shammi Kabab	435	25.0	25.8	25.8	3.6	180.0	3.4	890.0	58.00	0.29	0.21	7.4	3.7	1 Bowl
Shepherd's Pie	486	23.8	34.5	20.0	2.2	206.0	3.5	339.0	98.00	0.31	0.18	9.2	15	1 Bowl
Roast Chicken	297	25.3	21.8	0.0	0.0	18.0	2.0	334..0	166.0	0.13	0.20	10.0	0	1 Bowl
Chilli Chicken	464	27.3	35.5	8.8	1.9	46.0	3.1	222.0	135.0	0.35	0.31	10.9	50	1 Bowl
Chicken Sweet and Sour	420	27.0	33.3	3.1	0.8	39.0	2.7	270.0	181.0	0.28	0.35	10.6	30	1 Bowl
Chicken Korma	493	28.1	36.4	13.2	2.1	188.0	3.2	94.0	127.0	0.23	0.32	10.2	11	1 Bowl
Bengal Fish Curry	296	18.4	17.5	16.3	1.6	731.0	1.7	22.4	7.00	0.14	0.11	1.1	35	1 Bowl
Tandoori Fish	174	25.7	6.8	2.4	0.0	7.0	3.1	10.0	11.00	0.01	0.02	0.0	0	1 Bowl
Fried Fish with Chips	443	26.3	26.2	25.4	2.3	307.0	3.3	165.0	94.00	0.09	0.11	0.9	9	1 Bowl
Fish in Coconut Milk	371	27.0	17.1	27.2	3.2	150.0	3.2	4.0	1.00	0.06	0.03	0.5	7	1 Bowl
Prawn Curry	342	30.1	19.9	10.5	2.7	509.0	9.3	3.0	0.80	0.05	0.18	7.5	4	1 Bowl
Crispy Baked Fish	390	32.3	15.1	31.2	4.1	461.0	4.1	496.0	153.00	0.13	0.13	1.3	16	1 Bowl
VEGETABLES AND PANEER														
Nutri Nugget Sweet and Sour	252	12.8	18.3	9.1	8.0	22.0	1.0	180.0	79.00	0.16	0.16	1.0	30	1 Bowl
Nutri Nugget Korma	347	14.2	23.4	20.0	2.1	217.0	1.4	28.0	12.00	0.11	0.13	0.6	11	1 Bowl
Mashrooom Matar	239	10.3	10.8	25.2	2.8	68.5	3.3	298.0	75.00	0.41	0.30	4.1	31	1 Bowl
Nutri Nugget Matar	283	18.4	10.5	28.6	2.8	68.5	2.4	298.0	75.00	0.35	0.05	1.0	29	1 Bowl
Palak Mushroom	161	5.2	10.6	11.3	7.7	149.0	2.9	708.0	2177.00	0.17	0.59	3.0	60	1 Bowl
Vegetable Nargisi Kofta Curry	394	17.1	33.1	6.8	6.7	188.0	2.0	146.0	103.0	0.30	0.06	2.6	43	1 Bowl
Paneer Makhani	659	9.5	61.3	17.4	3.0	305.0	2.9	2046.0	580.0	0.36	0.19	1.3	64	1 Bowl
Pea Potato Curry	267	8.9	10.3	34.6	4.1	62.8	2.2	165.0	41.00	0.37	0.03	1.7	30	1 Bowl
Pea Paneer Curry	351	17.3	20.8	23.6	2.8	162.0	2.0	153.0	94.00	0.36	0.04	1.1	23	1 Bowl
Pea Curry	311	15.3	10.3	39.2	4.5	78.0	3.5	236.0	60.00	0.60	0.04	2.0	30	1 Bowl
Pea/Cholia Vadi Curry	309	11.7	15.3	31.0	3.7	68.0	2.8	195.0	49.00	0.50	0.03	1.5	26	1 Bowl
Potato Curry	221	2.5	10.2	29.9	3.6	47.7	1.0	94.0	23.60	0.17	0.03	1.5	29	1 Bowl
Egg Curry	314	15.5	17.6	23.3	2.8	85.0	2.8	483.0	237.00	0.38	0.25	1.2	21	1 Bowl
Dahi Aloo	260	4.2	12.2	33.4	3.9	119.0	1.0	169.0	58.00	0.22	0.12	1.8	35	1 Bowl
Dum Aloo	291	2.1	21.1	23.2	2.5	41.5	0.5	29.0	8.00	0.11	0.03	1.2	17	1 Bowl
Ghia Kofta Curry	273	3.6	20.9	17.5	2.9	70.0	1.9	122.0	31.00	0.17	0.05	0.9	15	1 Bowl
Potato Kofta Curry	290	2.9	15.3	35.1	4.1	51.0	1.3	101.0	25.00	0.19	0.03	1.80	33	1 Bowl

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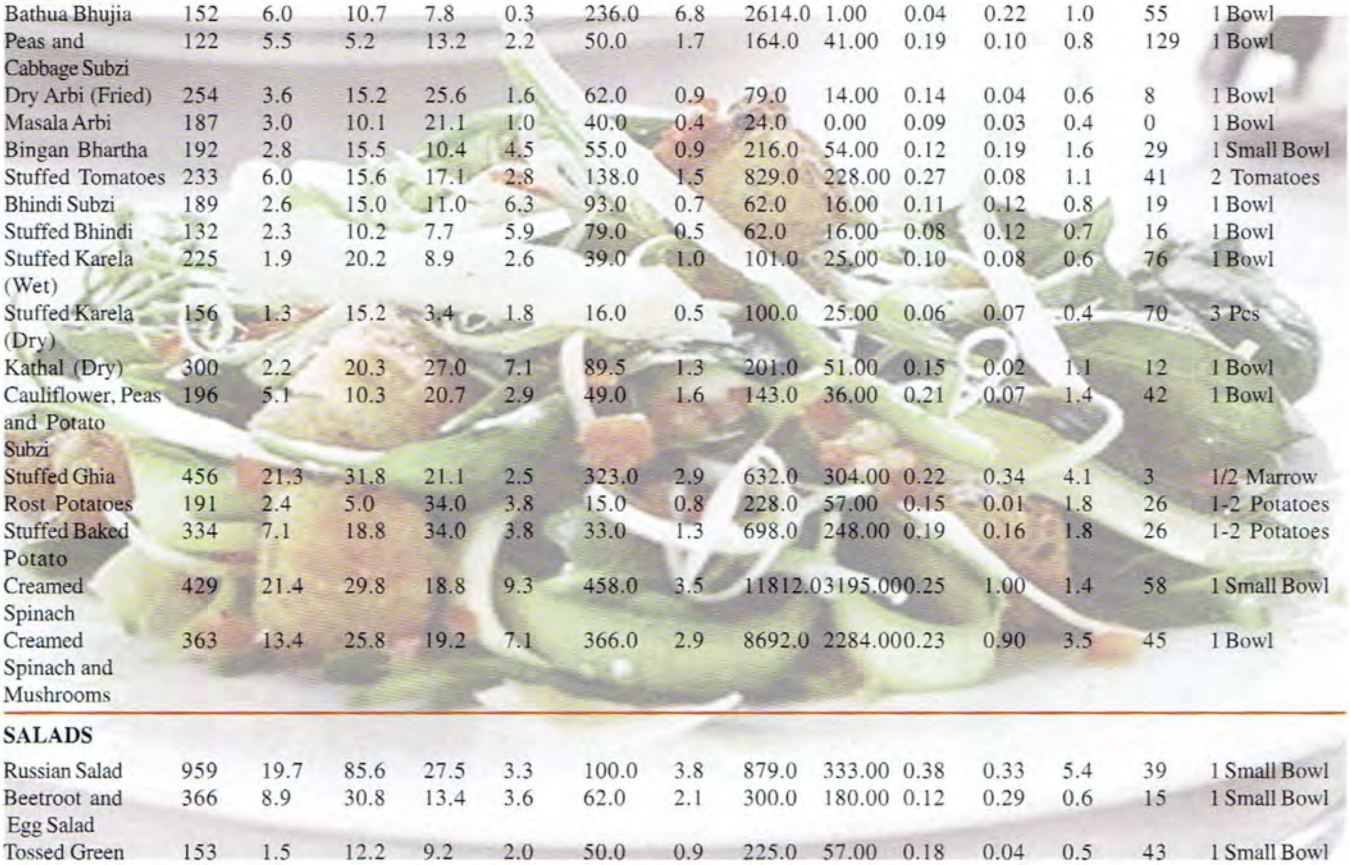

	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
Cabbage Kofta Curry	297	5.9	21.0	21.0	4.0	85.0	2.1	210.0	52.00	0.20	0.14	1.1	136	1 Bowl
Paneer Kofta Curry	383	11.2	30.7	15.5	1.5	149.0	0.9	74.0	74.00	0.13	0.04	0.6	15	1 Bowl
Aloo Methi	221	5.8	11.0	24.7	4.1	404.0	2.5	2361.0	590.00	0.12	0.32	1.8	66	1 Bowl
Gajar Methi	180	5.2	11.0	15.0	3.5	460.0	2.8	3854.0	963.00	0.08	0.33	1.3	55	1 Bowl
Sarson Ka Saag	157	8.5	6.7	15.7	3.2	200.0	25.0	6734.0	1682.00	0.22	0.14	0.5	66	1 Bowl
Palak Paneer	240	11.2	17.5	9.4	7.7	228.0	2.1	8545.0	2180.00	0.16	0.43	1.1	59	1 Bowl
Palak Aloo	230	5.1	11.3	27.0	9.7	153.0	2.5	8564.0	2141.00	0.20	0.40	2.0	72	1 Bowl
Mooli Bhujia	144	4.6	10.6	7.6	2.3	294.0	0.8	5300.0	2.00	0.23	0.49	1.2	91	1 Bowl
Bathua Bhujia	152	6.0	10.7	7.8	0.3	236.0	6.8	2614.0	1.00	0.04	0.22	1.0	55	1 Bowl
Peas and Cabbage Subzi	122	5.5	5.2	13.2	2.2	50.0	1.7	164.0	41.00	0.19	0.10	0.8	129	1 Bowl
Dry Arbi (Fried)	254	3.6	15.2	25.6	1.6	62.0	0.9	79.0	14.00	0.14	0.04	0.6	8	1 Bowl
Masala Arbi	187	3.0	10.1	21.1	1.0	40.0	0.4	24.0	0.00	0.09	0.03	0.4	0	1 Bowl
Bingan Bhartha	192	2.8	15.5	10.4	4.5	55.0	0.9	216.0	54.00	0.12	0.19	1.6	29	1 Small Bowl
Stuffed Tomatoes	233	6.0	15.6	17.1	2.8	138.0	1.5	829.0	228.00	0.27	0.08	1.1	41	2 Tomatoes
Bhindi Subzi	189	2.6	15.0	11.0	6.3	93.0	0.7	62.0	16.00	0.11	0.12	0.8	19	1 Bowl
Stuffed Bhindi	132	2.3	10.2	7.7	5.9	79.0	0.5	62.0	16.00	0.08	0.12	0.7	16	1 Bowl
Stuffed Karela (Wet)	225	1.9	20.2	8.9	2.6	39.0	1.0	101.0	25.00	0.10	0.08	0.6	76	1 Bowl
Stuffed Karela (Dry)	156	1.3	15.2	3.4	1.8	16.0	0.5	100.0	25.00	0.06	0.07	0.4	70	3 Pcs
Kathal (Dry)	300	2.2	20.3	27.0	7.1	89.5	1.3	201.0	51.00	0.15	0.02	1.1	12	1 Bowl
Cauliflower, Peas and Potato Subzi	196	5.1	10.3	20.7	2.9	49.0	1.6	143.0	36.00	0.21	0.07	1.4	42	1 Bowl
Stuffed Ghia	456	21.3	31.8	21.1	2.5	323.0	2.9	632.0	304.00	0.22	0.34	4.1	3	1/2 Marrow
Rost Potatoes	191	2.4	5.0	34.0	3.8	15.0	0.8	228.0	57.00	0.15	0.01	1.8	26	1-2 Potatoes
Stuffed Baked Potato	334	7.1	18.8	34.0	3.8	33.0	1.3	698.0	248.00	0.19	0.16	1.8	26	1-2 Potatoes
Creamed Spinach	429	21.4	29.8	18.8	9.3	458.0	3.5	11812.0	3195.00	0.25	1.00	1.4	58	1 Small Bowl
Creamed Spinach and Mushrooms	363	13.4	25.8	19.2	7.1	366.0	2.9	8692.0	2284.00	0.23	0.90	3.5	45	1 Bowl
SALADS														
Russian Salad	959	19.7	85.6	27.5	3.3	100.0	3.8	879.0	333.00	0.38	0.33	5.4	39	1 Small Bowl
Beetroot and Egg Salad	366	8.9	30.8	13.4	3.6	62.0	2.1	300.0	180.00	0.12	0.29	0.6	15	1 Small Bowl
Tossed Green Salad	153	1.5	12.2	9.2	2.0	50.0	0.9	225.0	57.00	0.18	0.04	0.5	43	1 Small Bowl
Cucumber and Yogurt Salad	29	1.3	1.3	2.9	1.0	53.0	0.5	10.0	4.00	0.04	0.05	0.2	6	1 Small Bowl
French Dressing	722	0.0	80.0	0.4	0.0	1.0	0.0	0.0	0.00	0.00	0.00	0.01	1	3/4 Cup
Mayonnaise	1220	7.1	131.8	1.3	0.0	56.0	1.4	380.0	229.00	0.08	0.26	0.0	4	1 Cup
Mayonnaise without Eggs	886	7.7	90.1	11.0	0.0	288.0	0.5	139.0	36.00	1.20	0.46	0.3	6	1 Cup
RAITAS														
Tomato Onion Raita	79	3.7	4.1	6.7	0.6	173.0	0.5	120.0	36.00	0.10	0.18	0.3	11	1 Bowl
Cucumber Raita	68	3.3	4.1	4.5	0.8	155.0	0.6	32.0	14.00	0.07	0.16	0.2	5	1 Bowl
Carrot Raita	85	3.6	4.1	8.3	0.9	189.0	0.7	977.0	250.00	0.07	0.17	0.4	3	1 Bowl

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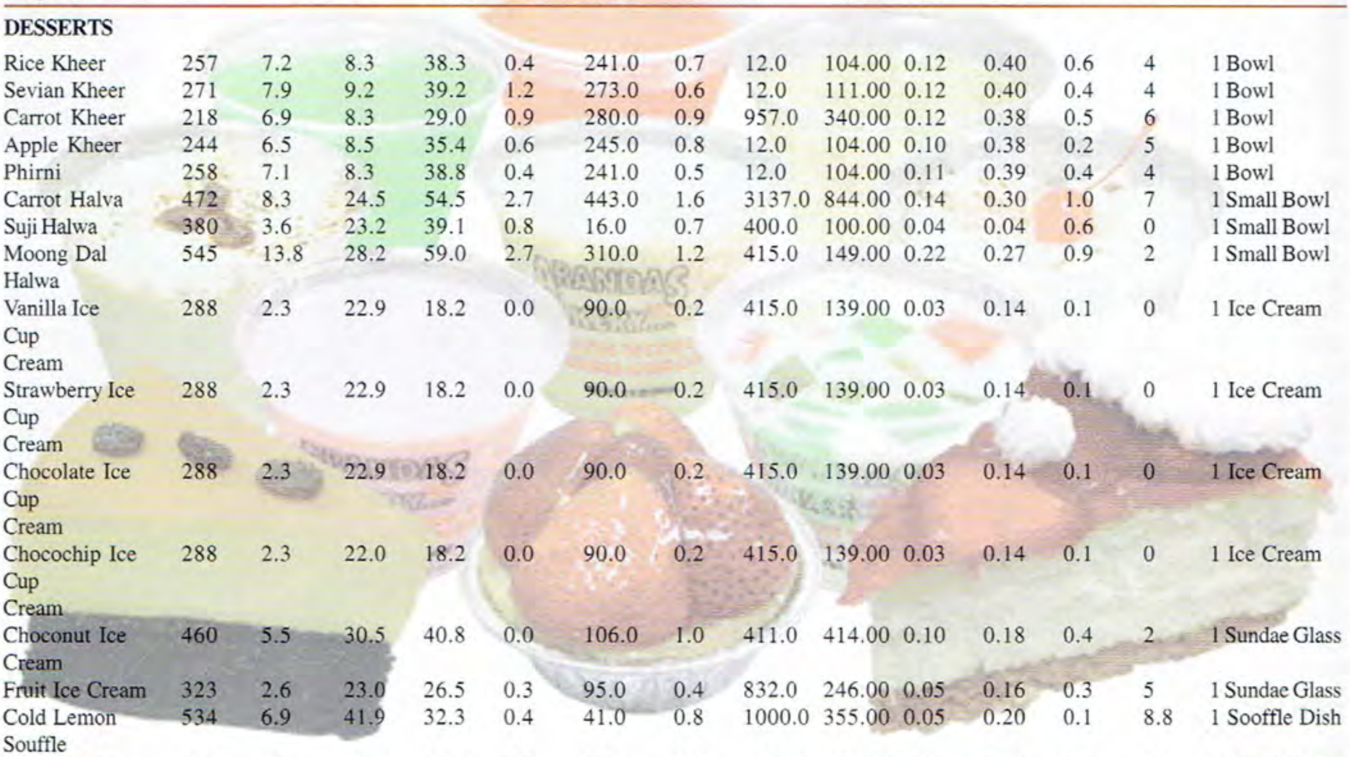
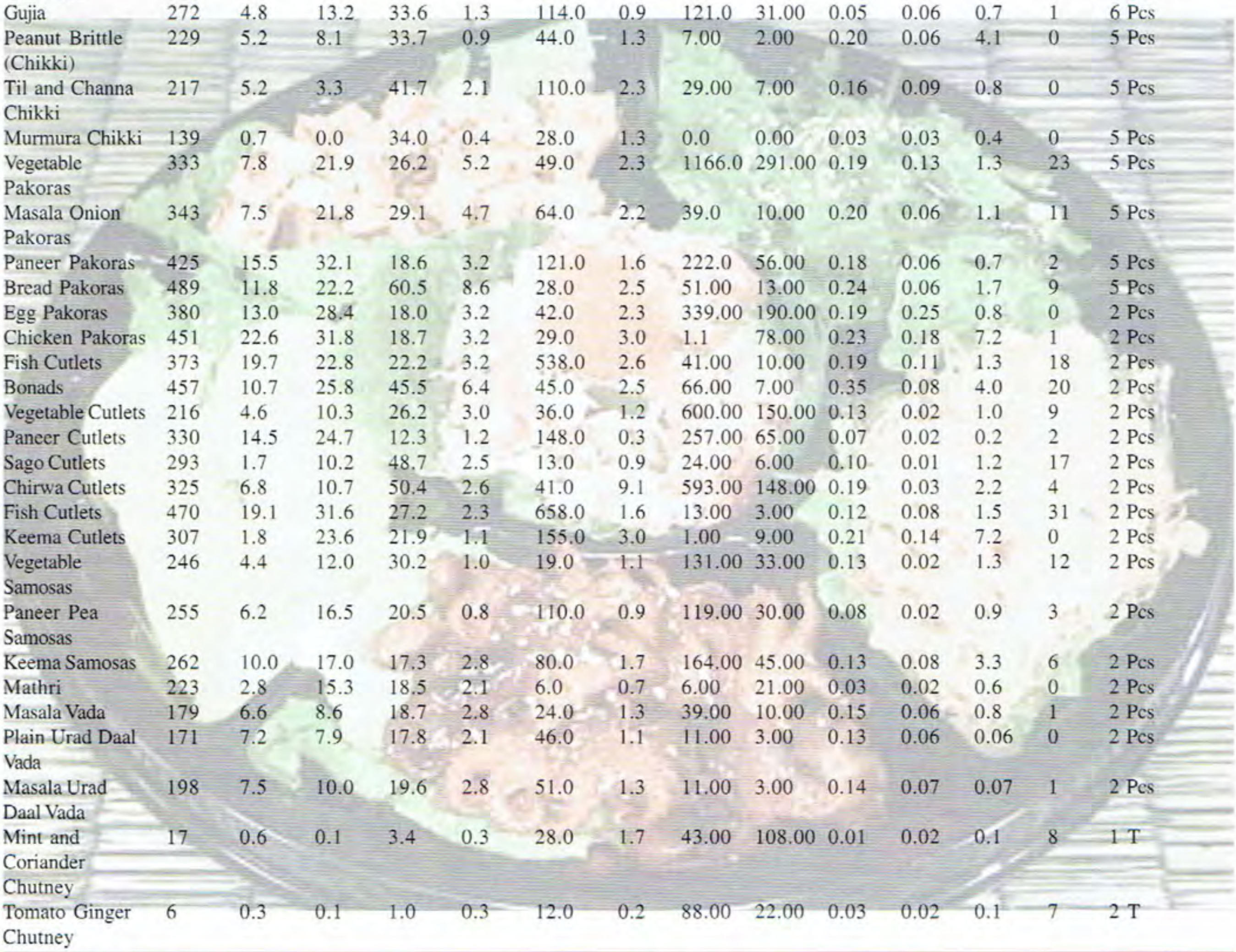


	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
Ghia Raita	67	3.2	4.1	4.3	0.3	159.0	0.4	32.0	14.00	0.06	0.17	0.2	1	1 Bowl
Potato Raita	110	3.9	4.1	14.3	1.3	154.0	0.5	44.0	17.00	0.10	0.17	0.7	10	1 Bowl
Spinach Raita	143	7.7	8.7	8.5	4.9	231.0	1.6	5616.0	1410.00	0.17	0.43	2.6	29	1 Bowl
Bathua Raita	147	9.3	8.4	8.5	0.4	308.0	4.7	1776.0	15.00	0.15	0.31	2.7	36	1 Bowl
Sprouted Green Gram Raita	105	5.8	4.1	11.2	1.2	166.0	0.7	37.0	15.00	0.11	0.18	0.4	3	1 Bowl
Boondi Raita	224	7.3	15.0	15.0	2.1	160.0	1.3	58.0	21.00	0.15	0.20	0.6	1	1 Bowl
Pineapple Raita	84	3.3	4.1	8.4	0.0	159.0	1.4	41.0	16.00	0.15	0.22	0.2	21	1 Bowl
Banana Raita	118	3.7	4.1	16.6	0.4	158.0	0.4	71.0	24.00	0.08	0.20	0.4	5	1 Bowl
Mango Raita	97	3.4	4.2	11.5	0.5	156.0	0.9	1404.0	357.00	0.09	0.20	0.6	9	1 Bowl
Grape Raita	91	3.4	4.2	10..0	1.4	159.0	0.5	33.0	14.00	0.06	0.17	0.2	2	1 Bowl
DESSERTS														
Rice Kheer	257	7.2	8.3	38.3	0.4	241.0	0.7	12.0	104.00	0.12	0.40	0.6	4	1 Bowl
Sevian Kheer	271	7.9	9.2	39.2	1.2	273.0	0.6	12.0	111.00	0.12	0.40	0.4	4	1 Bowl
Carrot Kheer	218	6.9	8.3	29.0	0.9	280.0	0.9	957.0	340.00	0.12	0.38	0.5	6	1 Bowl
Apple Kheer	244	6.5	8.5	35.4	0.6	245.0	0.8	12.0	104.00	0.10	0.38	0.2	5	1 Bowl
Phirni	258	7.1	8.3	38.8	0.4	241.0	0.5	12.0	104.00	0.11	0.39	0.4	4	1 Bowl
Carrot Halva	472	8.3	24.5	54.5	2.7	443.0	1.6	3137.0	844.00	0.14	0.30	1.0	7	1 Small Bowl
Suji Halwa	380	3.6	23.2	39.1	0.8	16.0	0.7	400.0	100.00	0.04	0.04	0.6	0	1 Small Bowl
Moong Dal Halwa	545	13.8	28.2	59.0	2.7	310.0	1.2	415.0	149.00	0.22	0.27	0.9	2	1 Small Bowl
Vanilla Ice Cup Cream	288	2.3	22.9	18.2	0.0	90.0	0.2	415.0	139.00	0.03	0.14	0.1	0	1 Ice Cream
Strawberry Ice Cup Cream	288	2.3	22.9	18.2	0.0	90.0	0.2	415.0	139.00	0.03	0.14	0.1	0	1 Ice Cream
Chocolate Ice Cup Cream	288	2.3	22.9	18.2	0.0	90.0	0.2	415.0	139.00	0.03	0.14	0.1	0	1 Ice Cream
Chocochip Ice Cup Cream	288	2.3	22.0	18.2	0.0	90.0	0.2	415.0	139.00	0.03	0.14	0.1	0	1 Ice Cream
Choconut Ice Cream	460	5.5	30.5	40.8	0.0	106.0	1.0	411.0	414.00	0.10	0.18	0.4	2	1 Sundae Glass
Fruit Ice Cream	323	2.6	23.0	26.5	0.3	95.0	0.4	832.0	246.00	0.05	0.16	0.3	5	1 Sundae Glass
Cold Lemon Souffle	534	6.9	41.9	32.3	0.4	41.0	0.8	1000.0	355.00	0.05	0.20	0.1	8.8	1 Sooffle Dish
Cold Orange Souffle	594	7.8	42.0	46.2	1.5	64.0	1.2	2656.0	769.00	0.05	0.20	0.1	45	1 Souffle Dish
Cold Pineappple Souffle	525	6.7	42.0	30.00	0.0	25.0	0.7	1000.0	355.00	0.00	0.20	0.1	0	1 Souffle Dish
Cold Vanila Souffle	536	7.3	42.7	30.6	0.0	57.0	0.7	1000.0	362.00	0.06	0.20	0.1	0.2	1 Souffle Dish
Cold Chocolate Souffle	536	7.3	42.7	30.6	0.0	57.0	0.7	1000.0	362.00	0.06	0.20	0.1	0.2	1 Souffle Dish
Bread and Butter Pudding	222	7.4	11.3	22.7	1.0	124.0	0.7	316.0	177.00	0.08	0.27	0.2	2	1 Small Plate
SNACKS														
Gulabjamuns	329	6.7	12.9	46.6	0.2	279.0	0.2	145.0	36.00	0.23	0.17	0.3	2	2 Pcs
Chenna Murkhi	275	9.3	10.6	35.6	0.0	110.0	0.0	186.0	47.00	0.06	0.02	0.0	2	10 Pcs
Plain Barfi	276	10.0	13.0	29.8	0.0	478.0	0.0	249.0	63.00	0.12	0.21	0.2	3	2 Pcs
Coconut Barfi	472	11.7	28.6	41.9	4.3	578.0	2.0	249.0	63.00	0.14	0.21	1.0	5	2 Pcs

Contd.

Contd.

	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
Besan Burfi	266	5.3	13.9	30.0	2.7	14.0	1.3	282.0	71.00	0.12	0.05	0.6	1	2 Pcs
Khoya Laddoo	375	10.7	22.2	33.0	1.8	441.0	1.4	206.0	52.00	0.14	0.21	0.8	3	2 Pcs
Besan Laddoo	286	5.8	16.4	28.7	2.8	15.0	1.4	332.0	83.00	0.13	0.05	0.7	1	2 Pcs
Suji Laddoo with Nuts	290	3.4	16.7	31.4	0.8	16.0	0.8	306.00	77.00	0.09	0.03	0.5	0	2 Pcs
Suji Laddoo with Khoya	355	5.6	20.1	38.0	1.9	125.0	0.6	312.00	78.00	0.07	0.07	0.5	1	2 Pcs
Til Laddo	158	3.2	7.2	20.0	0.0	256.0	2.0	10.0	3.00	0.18	0.07	0.8	0	6 Pcs
Gujia	272	4.8	13.2	33.6	1.3	114.0	0.9	121.0	31.00	0.05	0.06	0.7	1	6 Pcs
Peanut Brittle (Chikki)	229	5.2	8.1	33.7	0.9	44.0	1.3	7.00	2.00	0.20	0.06	4.1	0	5 Pcs
Til and Channa Chikki	217	5.2	3.3	41.7	2.1	110.0	2.3	29.00	7.00	0.16	0.09	0.8	0	5 Pcs
Murmura Chikki	139	0.7	0.0	34.0	0.4	28.0	1.3	0.0	0.00	0.03	0.03	0.4	0	5 Pcs
Vegetable Pakoras	333	7.8	21.9	26.2	5.2	49.0	2.3	1166.0	291.00	0.19	0.13	1.3	23	5 Pcs
Masala Onion Pakoras	343	7.5	21.8	29.1	4.7	64.0	2.2	39.0	10.00	0.20	0.06	1.1	11	5 Pcs
Paneer Pakoras	425	15.5	32.1	18.6	3.2	121.0	1.6	222.0	56.00	0.18	0.06	0.7	2	5 Pcs
Bread Pakoras	489	11.8	22.2	60.5	8.6	28.0	2.5	51.00	13.00	0.24	0.06	1.7	9	5 Pcs
Egg Pakoras	380	13.0	28.4	18.0	3.2	42.0	2.3	339.00	190.00	0.19	0.25	0.8	0	2 Pcs
Chicken Pakoras	451	22.6	31.8	18.7	3.2	29.0	3.0	1.1	78.00	0.23	0.18	7.2	1	2 Pcs
Fish Cutlets	373	19.7	22.8	22.2	3.2	538.0	2.6	41.00	10.00	0.19	0.11	1.3	18	2 Pcs
Bonads	457	10.7	25.8	45.5	6.4	45.0	2.5	66.00	7.00	0.35	0.08	4.0	20	2 Pcs
Vegetable Cutlets	216	4.6	10.3	26.2	3.0	36.0	1.2	600.00	150.00	0.13	0.02	1.0	9	2 Pcs
Paneer Cutlets	330	14.5	24.7	12.3	1.2	148.0	0.3	257.00	65.00	0.07	0.02	0.2	2	2 Pcs
Sago Cutlets	293	1.7	10.2	48.7	2.5	13.0	0.9	24.00	6.00	0.10	0.01	1.2	17	2 Pcs
Chirwa Cutlets	325	6.8	10.7	50.4	2.6	41.0	9.1	593.00	148.00	0.19	0.03	2.2	4	2 Pcs
Fish Cutlets	470	19.1	31.6	27.2	2.3	658.0	1.6	13.00	3.00	0.12	0.08	1.5	31	2 Pcs
Keema Cutlets	307	1.8	23.6	21.9	1.1	155.0	3.0	1.00	9.00	0.21	0.14	7.2	0	2 Pcs
Vegetable Samosas	246	4.4	12.0	30.2	1.0	19.0	1.1	131.00	33.00	0.13	0.02	1.3	12	2 Pcs
Paneer Pea Samosas	255	6.2	16.5	20.5	0.8	110.0	0.9	119.00	30.00	0.08	0.02	0.9	3	2 Pcs
Keema Samosas	262	10.0	17.0	17.3	2.8	80.0	1.7	164.00	45.00	0.13	0.08	3.3	6	2 Pcs
Mathri	223	2.8	15.3	18.5	2.1	6.0	0.7	6.00	21.00	0.03	0.02	0.6	0	2 Pcs
Masala Vada	179	6.6	8.6	18.7	2.8	24.0	1.3	39.00	10.00	0.15	0.06	0.8	1	2 Pcs
Plain Urad Daal Vada	171	7.2	7.9	17.8	2.1	46.0	1.1	11.00	3.00	0.13	0.06	0.06	0	2 Pcs
Masala Urad Daal Vada	198	7.5	10.0	19.6	2.8	51.0	1.3	11.00	3.00	0.14	0.07	0.07	1	2 Pcs
Mint and Coriander Chutney	17	0.6	0.1	3.4	0.3	28.0	1.7	43.00	108.00	0.01	0.02	0.1	8	1 T
Tomato Ginger Chutney	6	0.3	0.1	1.0	0.3	12.0	0.2	88.00	22.00	0.03	0.02	0.1	7	2 T
SANDWICHES														
Tomato and Cheese	268	7.2	11.8	33.3	4.6	88.0	0.9	460.0	145.00	0.09	0.06	0.6	11	2 Pcs
Tomato and Cucumber	231	5.2	8.6	33.1	4.9	28.0	1.0	460.0	115.00	0.10	0.02	0.6	12	2 Pcs
Tomato Grilled	313	9.6	15.0	34.8	5.0	160.0	1.2	530.0	192.00	0.12	0.12	0.6	16	4 Pcs
French Toast	443	14.6	27.6	34.0	4.6	144.0	1.7	1088.0	407.00	0.15	0.20	0.6	11	2 Pcs
Cheese Open	340	11.7	18.6	31.5	7.4	196.0	1.1	328.0	168.00	0.14	0.11	1.5	0	2 Pcs



Contd.

Contd.

	Calories (Kcal)	*Proteins (g)*	*Fats (g)*	*Carbo-hydrates (g)*	*Fiber (g)*	*Calcium (mg)*	*Iron (mg)*	*Caro-tene (mcg)*	*Retinol (mcg)*	*Vit B1 (mg)*	*Vit B2 (mg)*	*Niacin (mg)*	*Vit C (mg)*	*Serving Portion*
Danish Luncheon	350	16.2	17.5	31.9	4.6	42.0	2.1	620.0	260.00	0.24	0.22	1.2	3	2 Pcs
Chicken and Corn Open	340	15.6	15.0	35.6	5.5	18.0	1.8	362.0	124.0	0.12	0.11	4.6	3	2 Pcs
CAKES														
Sponge Cake	177	6.2	4.6	27.8	0.5	20.2	0.9	202.0	120.00	0.05	0.14	0.4	0	1 Pc
Sponge Chocolate Cake	156	5.5	4.5	23.3	0.3	18.8	0.7	201.0	120.00	0.05	0.14	0.3	0	1 Pc
Pineapple Pastry	279	7.1	13.2	32.9	0.6	23.1	1.0	390.0	176.00	0.06	0.16	0.5	0	1 Pastry
Chocolate Pastry	228	5.5	12.5	23.3	0.3	19.0	0.7	361.0	160.00	0.05	0.14	0.3	0	1 Pastry
Chocolate Cream Cake	223	3.1	13.1	23.2	0.4	9.0	0.5	526.0	158.00	0.03	0.06	0.3	0	1 Pc

Investigations

DIFFERENTIAL DIAGNOSIS FOR ALTERED BLOOD VALUES

1. SERUM PHOSPHORUS

Normal	Children: 4-7 mg/100 ml Adults: 3-4 mg/100 ml		
Elevated	Renal insufficiency Hypoparathyroidism Hypervitaminosis-D	**Decreased**	Hyperparathyroidism Rickets/osteomalacia Steatorrhea Antacid ingestion

2. SERUM TRIGLYCERIDE

Normal	Below 165 mg/100 ml		
Elevated	Hyperthyroidism Diabetes mellitus Nephrotic syndrome Biliary obstruction Primary hyperproteinemias	**Decreased**	Malabsorption Malnutrition Primary hypolipoproteinemias

3. SERUM BILIRUBIN

Normal total	0.3 to 1.1 mg/100 ml
Direct	0.1 to 0.4 mg/100 ml
Indirect	0.2 to 0.7 mg/100 ml

Rise of indirect serum bilirubin	In hemolytic diseases Gilbert's disease Acute/chronic hepatitis
Rise of total serum bilirubin	Biliary tract obstruction Cancer of head of pancreas Gallstones

Elevation of both conjugate and nonconjugate levels (with the conjugate levels more elevated)

Hepatic metastasis
Hepatitis
Lymphoma
Cirrhosis
Cholestasis secondary to drugs

4. SERUM CALCIUM

Normal	9.6 to 10.9 mg/100 ml		
Elevated	Hyperparathyroidism (20 mg)	**Decreased**	Hypoparathyroidism
			Osteomalacia/rickets
	Hypervitaminosis D (17 mg)		Malabsorption syndrome
	Multiple myeloma		
	Cushing's syndrome		

5. SERUM CHLORIDE

Normal	350 to 375 mg/100 ml		
Increased	Excessive salt in diet	**Decreased**	*Abnormal loss due*
	Overtreatment with saline solution		to vomiting and diarrhea
	Decreased excretion in urinary		Excessive sweating
	tract obstruction		
	Acute/chronic nephritis		
	Decompensated heart disease		

6. SERUM CHOLESTEROL

Normal	150-250 mg/100 ml		
Increased	Xanthomatosis	**Decreased**	Hyperthyroidism
	Pregnancy		Acute infections
	Alcohol and fatty diet		Anemia with malnutrition
	Myxedema		
	Diabetes mellitus		
	Obesity		
	Nephrotic syndrome		

7. SERUM ALBUMIN

Normal	3.5 to 5.5 gm/100 ml		
Increased	Hemoconcentration	**Decreased**	Malnutrition
	Shock		Starvation
	Dehydration		Glomerulonephritis
			Hepatic insufficiency
			Leukemia
			Other malignancies

8. SERUM GLOBULIN

Normal	1.5 to 3 gram/100 ml		
Increased	*Hepatic disease*	**Decreased**	Starvation with
	Multiple myeloma		malnutrition
	Bacterial and viral infection		Lymphatic leukemia
	Typhus and malaria		Agammaglobulinemia

9. NITROGEN COMPOUND

Normal	Net protein nitrogen	15 to 35 mg/100 ml	
	Blood urea nitrogen	10 to 40 mg/100 ml	
	Serum creatinine	0.7 to 1.5 mg/100 ml	
Increased	Nephritis	**Decreased**	Hepatic failure
	Acute renal failure		Nephrosis
	Urinary tract obstruction		Low protein diet
	Dehydration		
	Shock, congestive cardiac failure		
	Adrenal insufficiency.		

10. BLOOD UREA

Normal	10-15 mg/100 ml		
Increased	Impaired and renal function	**Decreased**	Liver failure
	Shock/dehydration		Malnutrition
	Diabetes		Impaired absorption
	Acute myocardial infection		In celliac disease
	Gout		Nephrotic syndrome
	Excessive protein intake		Overhydration

11. BLOOD URIC ACID

Normal	2.2 to 8 mg%		
Increased	Gout	**Decreased**	Aspirin
	Metastatic cancer		Sulfinpyrazone
	Starvation/shock		
	Alcoholism		
	Multiple myeloma		
	Diabetic ketosis		
	Leukemia		

12. SERUM ALKALINE PHOSPHATASE

Normal	5 to 13 KA or 2 to 5 Bodansky units per 100 ml.		
Increased	Severe osteomalacia	**Decreased**	Hypothyroidism
	Osteogenic sarcoma		Growth retardation
	Metastasis to bones		
	Paget's disease		
	Myeloid leukemia		
	Hyperthyroidism		
	Pregnancy		

13. SERUM AMYLASE

Normal	0.5 to 2 Bodansky units		
Increased	Acute pancreatitis	**Decreased**	Necrotising hepatitis
	Carcinoma of pancreas		Severe burns
	Perforated peptic ulcer		Toxemia of pregnancy
	Acute cholecystitis		
	Cirrhosis liver		
	Mumps		
	Renal failure		

14. SERUM IRON

Normal	75 mcg/100 ml.		
Increased	Hemochromatosis Aplastic anemia Hemosiderosis Hemolytic anemias Pernicious anemia	**Decreased**	Iron deficiency anemia Nephrosis Chronic renal insufficiency Paroxysmal nocturnal hematuria

15. SERUM FIBRINOGEN

Normal	0.2 to 0.4 gm/100 ml		
Increased	Rheumatic fever Arthritis Glomerulonephritis	**Decreased**	Eclampsia of pregnancy Severe anemia Typhoid Fibrinolysis Metastases from carcinoma of prostate Hepatic insufficiency

16. ESTROGENS

Premenopausal	women: 4-60 ng/dl		
Postmenopausal	women: 3-14 ng/dl		
Increased	Feminizing tumors Masculanizing tumor True precocious puberty Liver cirrhosis Corpus luteum cyst	**Decreased**	Turner's syndrome Menopausal Hypothalamic disorder Anovulatory bleeding Inadequate luteal phase

17. FASTING BLOOD SUGAR

Normal	60 to 100 mg percent		
Increased	Diabetes Cushing's disease Acute stress Pheochromocytoma Hyperthyroidism Pancreatitis Chronic liver disease Chronic malnutrition	**Decreased**	Over dose of insulin Addison's disease Bacterial sepsis Islet cell carcinoma Hepatic necrosis Hypothyroidism Psychogenic causes

18. SGPT

Normal	5-35 Sigma frankel/ml, 4-24 IU/liter
Increased	Hepatocellular disease Active cirrhosis Metastatic liver tumor Obstructive liver tumor Obstructive jaundice Liver congestion Pancreatitis Hepatic injury in myocardial infarction

19. SGOT

Normal	5-40 Sigma Frankel u/ml, 0-36 IU/liter		
Increased	Myocardial infarction Liver diseases Acute pancreatitis Acute hemolytic anemia Acute renal disease Severe burns Recent brain trauma	**Decreased**	Beri beri Uncontrolled diabetes

20. LACTIC ACID DEHYDROGENASE (LDH)

Normal	63-155 units		
Increased	Acute myocardial infarction Acute leukemia Hepatic disease Extensive cancer Shock and anoxia	**Decreased**	Good response to cancer

21. HDL CHOLESTEROL

Normal	44 mg/dl in men and 55 mg/dl in women		
Increased	Chronic liver disorder Increased physical activity Moderate intake of alcohol	**Decreased**	Smokers High risk patients of MI

22. THYROXINE TOTAL T_4

Normal	5-12.5 ug/dl		
Increased	Hyperthyroidism Acute thyroiditis Subacute thyroiditis Hepatitis	**Decreased**	Cretinism Myxedema Simmonds' disease Hypothyroidism Nephrosis

23. TRIIODOTHYRONINE (T_3)

Normal	110-230 ng/100 ml.		
Increased	Hyperthyroidism T_3 thyrotoxicosis Acute thyroiditis Idiopathic TBG elevation	**Decreased**	Hypothyroidism Starvation Acute illness Idiopathic TBG decrease

24. CAROTENE (VIT. A RETINOL)

Normal	50-300 ug/100 ml		
Increased	Myxedema Diabetes mellitus Hypothyroidism Chronic nephritis Hyperlipidemia	**Decreased**	Fat malabsorption syndrome Streatorrhea Febrile illness Liver disease Poor dietary intake

25. LIPASE

Normal 4-24 IU/dL

Increased
Pancreatitis
Pancreatic carcinoma
Acute cholecystitis
Cirrhosis
Severe renal disease
Obstruction of pancreatic duct.

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26. CALCIUM

Normal Calcium values
1 week to 2 years — 9.0-11.0 mg/dl
2 to 18 years — 8.8-10.2 mg/dl
Adult — 8.6-mg/dl

Hypercalcemia
Increased
Metastatic bone cancer from lung, kidney, breast, liver and pancreas
Multiple myeloma
Sarcoidosis
Hyperthyroidism
Prolonged rest in bed as in burns/fracture
Excassive intake of vitamin-D

Calcium levels above 13 mg/dl may cause
(i) Cardiotoxicity
(ii) Arrhythmias

Hypocalcium
Decreased
Excessive IV fluids will decrease albumin and thus calcium because 50% calcium is bound to proteins.
Malabsorption due to sprue, pancreatic dysfunction
Osteomalacia
Renal failure
Vitamin D deficiency

Calcium below 6 mg/dl may cause
(i) Tetany
(ii) Convulsions

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27. CHLORIDE

Normal chloride values
New born 98–113 mmol/L
Adult 98–106 mmol/L

Chloride maintains
Cellular integrity
Acid-base balance
Water balance

Increased blood chloride level
Dehydration
Diabetes incipidus
Hyperparathyroidism
Hyperventilation
Metabolic disorder
Excessive saline infusions

Decreased blood chloride levels
Burns
Severe vomiting
Diabetes
Overhydration
Diuretic therapy

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28. MAGNESIUM

Child — 1.4-1.7 mEq/L
Adult — 1.3-2.1 mEq/L

Increased blood magnesium levels
Dehydration
Renal failure

Decreased blood magnesium levels
Severe burns
Excessive loss of body fluids
Hyperthyroidism

Severe diabetic acidosis	Colitis
Use of antacids containing magnesium	Prolonged gastric drainage

- Magnesium deficiency may cause muscle tremors, hyperactive deep reflexes lethargy, insomnia and hypocalcemia
- Too much magnesium will cause lethargy, slurred speech, weak/absent deep tendon reflexes, hypotension drowsiness and respiratory depression.

29. POTASSIUM

Normal values 1-18 years—3.4-4.7 mEq/L
Adult—3.5-5.3 mEq/L

Potassium is needed in nerve conduction muscle function and acid-base balance. It controls rate and force of contraction of heart with calcium.

Increased potassium levels	**Decreased potassium levels**
Cell damage in burns	Starvation
Acidosis	Severe burns
Uncontrolled diabetes	Respiratory alkalosis
Renal failure	Renal tubular acidosis
Accident/Surgery	Draining wounds

30. SODIUM

Normal values 135-145 mEq/L
Adult

It maintains acid-base balance and transmit nerve impulses

Increased sodium level	**Decreased level of sodium**
Dehydration	Severe burns
Diabetes insipidus	Excessive fluid loss
Coma	Excessive IV glucose
Anabolic steroids	Severe nephritis
High triglycerides	Diabetic acidosis
	Large amount of water orally

31. LEUKOCYTOSIS

An increase of WBC above 10,000/cu mm

Increased	**Decreased**
Any infection	Viral infection
Hemorrhage	Hypersplenism
Trauma/tissue injury	Bone marrow depression due to drugs
Serum sickness	
Malignancy	
Leukemia	
Tissue necrosis	

32. EOSINOPHILIA

Normal absolute value 50-250/cu.mm
Eosinophilia—increase more than 5% or more than 500.

Increased eosinophil	**Decreased eosinophils**
Allergies	Infectious mononucleosis
Parasitic diseases	Hypersplenism
Lung and bone cancer	Congestive cardiac failure
Chronic skin infections	Cushing's syndrome
Hodgkin's disease	Aplastic anemia
	Use of ACTH

33. LYMPHOCYTES

Normal	20-40% of total leukocyte count.		
Increased	Upper respiratory infections Viral diseases mumps etc Bacterial infections, i.e. TB etc. Hypothyroidism Lymphocytic leukemia	**Decreased**	Hodgkin's disease LE After ACTH After burns and trauma Chronic uremia

34. BLEEDING TIME

Normal	3-10 minutes
Increased	Thrombocytopenia Platelet dysfunction syndrome Severe liver disease Leukemia Aplastic anemia

35. PROTHROMBIN TIME

Normal	11-16 seconds
Increased	Prothrombin deficiency Vit K deficiency Hemorrhagic diseases Liver disease Biliary obstruction Hypervitaminosis A

36. PLATELET COUNT

Normal	150000-350000 per cmm.		
Increased	Cancer Chronic leukemia Polycythemia vera Splenectomy Heart disease Rheumatoid arthritis	**Decreased**	Thrombocytopenic purpura Pernicious anemia After massive blood transfusion Allergic condition Pneumonia Infection

37. MCV

Normal	83-103 cu m/red cell		
Increased	Liver disease Alcoholism Sprue Deficiency of folate or B_{12}	**Decreased**	Iron deficiency anemia Pernicious anemia Thalassemia Chlorosis

38. MCHC

Normal	32-36%		
Increased	spherocytosis	**Decreased**	Iron deficiency Macrocytic anemia Pyridoxine responsive anemia Thalassemia

URINE ANALYSIS

Specific Gravity

Normal	—1.010 to 1.025
Concentrated urine	—1.025 +
Diluted urine	—1.001 to 1.010

Specific gravity increases if excretion of urine decreases

Low Specific Gravity

- Diabetes insipidus
- Glomerulonephritis and pyelonephritis
- Severe renal damage

Increased Specific Gravity

- Diabetes mellitus or nephrosis
- Excessive water loss, i.e. sweating, fever, vomiting or diarrhea

Interfering Factors

- Specific gravity is highest in the first morning specimen
- Drug causing false positive elevation are dextran and radio-opaque contrast media

Color of Urine

- Colorless urine
    - Large fluid intake
    - Untreated diabetes mellitus
    - Diuretic therapy
    - Reduction in perspiration
    - Alcohol ingestion
    - Nervousness
- Orange colored urine
    - Concentrated urine
    - Excessive sweating
    - Restricted fluid intake
    - Fever
- Red or reddish dark brown
    - Hemoglobinuria
    - Myoglobin
    - Porphyrias
- Brown black
    - Lysol poisoning

Yellow foam due to biliverdin bile pigment, green foam, also is due to biliverdin bile pigment.

Interfering Factors

- Darkens on standing
- Beet will turn urine maroon red, rhubarb to red
- Orange may be due to pyridium, amidopyrin
- Orange yellow due to salicylasulfapyridine
- Rust yellow due to sulfonamides, nitrofurantoin
- Dark colored due to iron salts

Odor

- Sweet smell of acetone can be recognized in diabetic ketosis
- Heavy infected urine has unpleasant odor
- Long standing gives ammonia flavor

Turbidity

- Fresh urine is clear
- Urine becomes turbid due to urinary tract infection
- Urine may be cloudy due to the RBCs WBCs or bacteria

Interfering Factors

- After consumption of food, urate or phosphates may produce cloudiness
- Vaginal contamination from female patients is a common cause of turbidity
- Greasy cloudiness may be caused by large amounts of fat
- After refrigeration it will develop hazziness

Hemoglobinuria is found in:

- Extensive burns and crushing injuries
- Transfusion reactions to incompatible blood
- Febrile intoxication
- Malaria
- Hemolytic anemias

Drugs Which may Cause Positive Result

- Bacitracin
- Coumarin
- Aspirin

Interfering Factors

- High doses of vitamin C may give false negative result.

- Drugs causing false positive results are bromide, copper, iodides and oxidizing agents

Protein Normal Values 2-8 mg/dl

Proteinuria due to Kidney Diseases

- TB and cancer of kidney
- Polycystic kidney
- Nephrosis
- Nephritis
- Ascites

Proteinuria due to Non-renal Causes

- Fever
- Coumarin
- Severe anemias
- Toxemia
- Trauma
- Aspirin

Interfering Factors

- *Transitory proteinuria may be caused by:*
    - Violent exercise
    - Cold bath
    - Severe emotional stress
- *Increased protein in urine occurs:*
    - After eating large amount of protein
    - In newborn infants
    - In pregnancy or following delivery
    - In premenstrual state

Sugar Normal Values 100 mg/24 Hours

- Increased glucose in urine found in diabetes mellitus, brain injury, myocardial infarction

Interfering Factors

- Pregnancy and lactation may cause false positive
- Ascorbic acid, creatinine, streptomycin may cause false positive
- Stress, excitement, testing after heavy meal may give positive result

Ketone Bodies Normal Values—Negative

- Ketosis and ketonuria may occur whenever increased amounts of fats are metabolised, carbohydrate intake is restricted or diet is rich in fat
- Ketone bodies in urine appears first than blood

Ketonuria is Associated

- Fever
- Prolonged vomiting
- Fasting, starvation
- Gastrointestinal disturbances
- Anorexia

Drugs that may Cause False Positive

- Levodopa
- Pyridium
- Paraldehyde
- Insulin
- Ether

Red Cell Cast

- *These may be the only manifestations of:*
    - Acute glomerulonephritis
    - Collagen disease
    - Renal infarction
    - Endocarditis
- *Increased red cells occur in:*
    - Pyelonephritis
    - Trauma to the kidney
    - Lupus vulgaris
    - TB and malignancy
    - Renal stones
    - Hemophilia
    - Cystitis
- Red cells in excess of WBCs indicate bleeding into the urinary tract and in:
    - Trauma
    - Anticoagulant therapy
    - Thrombocytopenia
    - Aspirin
    - Tumor

Interfering Factors

- Increased number of RBCs can be found following exercise, traumatic catheterization, passage of stone or contamination by menstrual fluid

White Blood Cells

- Large number of white cells indicate bacterial infection urinary in tract
- If infection is in kidney there may be associated cellular or granular casts, bacteria epithelial cells and few RBCs

White Blood Cells and Casts

- Pyelonephritis
- Acute glomerulonephritis
- Interstitial inflammation of kidney

Epithelial Cells and Casts

- Renal epithelial cell casts are formed by cast of tubular cells, hence occasional renal epithelial cells are found. More are found in:
- Amyloidosis
- Poisoning from heavy metals

Hyaline Casts

Occasional hyaline casts per low power are found. These are clear, colorless casts formed when protein within the tubules precipitates and gels. Hyaline casts may be a temporary phenomenon due to:

- When large number of hyaline casts appear in urine along with heavy proteinuria, fine granular casts or fat droplet, nephrotic syndrome may be suggested.

Granular Casts

Occasional granular casts are found. But excessive may be found in:

- Acute tubular necrosis
- Pyelonephritis
- Chronic lead poisoning
- Advanced glomerulonephritis
- Malignant nephrosclerosis
- Chronic lead poisoning

Waxy Casts

Waxy costs found in:

- Chronic renal disease
- Tubular inflammation and degeneration

STOOL

Diarrhea mixed with mucus and blood is associated with:

- Typhus
- Amebiasis
- Typhoid
- Large bowel cancer
- Cholera

Diarrhea with mucus and pus mixed is due to:

- Ulcerative colitis
- Shigellosis
- Regional enteritis
- Salmonellosis
- In obstruction of common bile duct the fat gives a putty like appearance
- In sprue and celiac disease the stool resembles aluminium
- In cystic fibrosis the increase of neutral fat gives a greasy butter stool appearance

Alteration in shape and size

- Narrow ribbon like stool suggests spastic bowel, rectal narrowing, decreased elasticity or partial obstruction
- Excessive hard stool due to increased absorption of fluids, constipation
- Very large caliber stool indicate dilatation of viscus
- Small, round, hard stool accompany habitual, moderate constipation

Color of feces

- Yellow to yellow green diarrhea during breastfeed
- Green color occurs in chlorophyll rich vegetables
- Black color may be due to iron charcoal and bismuth
- Light colored stool with little odor may be due to diets high in milk and low in meat
- Clay like color may be due to excessive fat
- Red color may be due to beets

Certain Color Changes due to Drugs

Black iron salts, Charcoal
Green Mercurous chloride, Calomel
Green to blue Diathiazanine
Yellow Santonin
Yellow to brown Senna
Orange red Phenazopyridine

Occult Blood in Stool

- Meat in diet can give false positive test
- Vitamin C more than 500 mg may give false negative test
- Drugs which may cause false positive test
  - Boric acid
  - Bromides
  - Colchicine
  - Iodine
  - Iron
  - Oxidizing agent

DRUGS

Drugs Affecting Color of Stool

Black	Iron salts, bismuth, charcoal
Green	Indomethacin
Greenish blue	Diathiazanine
Red	Tetracycline syrups
Yellow	Santonin
Yellow-brown	Senna
Whitish	Antacids
Orange red	Phenazopyridine
Pink to black	Anticoagulant

Drugs Changing Color of Urine

Chloroquine	Brown
Furazolidine	Brown
Indomethacin	Green
Methyldopa	Red darkens on standing
Metronidazole	Dark brown
Phenacetin	Dark brown
Primaquine	Rust yellow
Mepacrine	Deep yellow
Rhubarb	Yellow brown
Riboflavin	Yellow
Rifampicin	Red orange
Salicylates	Pink red
Sulphonamides	Rust yellow

Drugs Causing Hyperglycemia

- Corticosteroid
- Indomethacin
- Isoniazid

Drugs causing False Positive Test by Reducing with Benedict's Solution

- Acetylsalicylic acid
- Chloral hydrate
- Streptomycin
- PAS
- Nitrofurantoin
- Sulphonamides

Drugs causing Urine Urobilinogen

- Aminosalicylic acid
- Cascara
- Phenothiazines
- Antipyrine
- Chlorpromazine
- Sulfonamides

Drugs that may cause Positive Test for Urine Porphyrins

- Acriflavin
- Sulfamethoxazole
- Phenazopyridine
- Tetracycline

Drugs which may Precipitate Porphyria

- Antipyretics
- Barbiturates
- Sulfonamides

Drugs which cause Increased Urine Calcium

- Anabolics/androgens
- Corticosteroids
- Vitamin D
- Parathyroid injection

Drugs Affecting Rate of ESR

- Increased ESR
  - Dextran
  - Vitamin A
  - Methyldopa
  - Theophylline
- Decreased ESR
  - Acetylsalicylic acid
  - Alcohol
  - Dextran
  - Streptokinase

Drugs causing Increased Values of Alkaline Phosphatase

- Albumin
- Colchicine
- Isoniazid
- Rifampicin
- Sulfonamides
- Anabolics
- Ergosterol
- PAS
- Tolbutamide
- methyl testosterone

Drugs Increasing Values of Amylase

- ACTH
- Corticosteroids
- Histamine
- Tetracycline
- Chlorides
- Diuretics
- Methylcholine
- Sulfamethazole

Drugs Increasing Serum Bilirubin

- Acetazolamide
- Anabolics
- Chloroquine
- Diazepam
- Papaverine
- Vitamin A
- PAS
- Barbiturates
- Dextran
- Erythromycin
- Phenacetamine
- Rifampicin

Drugs Increasing Blood Urea Level

- Alkaline antacid
- Vitamin C
- Bacitracin
- Arsenicals

- Chloral hydrate
- Gentamicin
- Sulfonamides
- Dextran
- Salicylates

Drugs causing Increased Values of Cholesterol

- ACTH
- Aspirin
- Iodides
- Progestin
- Vitamin A and D
- Androgens
- Chlorpromazine
- Phenothiazines
- Salicylates

Drugs Increasing Values of T_4

- Diethylstilbestrol
- Oral contraceptives
- Sulfamethoxazole
- Estrogen
- Progestine

Drugs Increasing Values of T_3

- Anabolic agents
- Anticoagulants
- Phenylbutazone
- Androgens
- Estrogens
- Salicylates

Drugs Increasing Values of Uric Acid

- Ampicillin
- Buscalfan
- Dextran
- Ethambutol
- Nicotinic acid
- Phenothiazines
- Diuretics
- Aspirin
- Coffee
- Diuretics
- Methyldopa
- Penicillin
- Rifampicin

Drugs Increasing Values of 17 Ketosteroids

- Androgenic steroid
- Chlorpromazine
- Erythyromycin
- Testosterone
- Nalidixic acid
- Ascorbic acid
- Digotoxin
- Quinine
- Morphine

Drugs causing Pregnancy Test Positive

- Aspirin
- Phenothiazines
- Fluphenazine
- Chlorpromazine
- Gravindex

Drugs causing Positive Result for Occult Blood in Stool

- Boric acid
- Cholchicine
- Iron
- Bromide
- Iodides
- Pyridium

Drugs Increasing Bleeding Time

- Acetyl salicylic acid
- Streptokinase
- Dextran
- Alcohol derivatives

Drugs Resulting in Decrease of Leukocyte Count

- Chloramphenicol
- Diazepam
- Methyldopa
- Penicillin
- Sulfonamide
- Amidopyrine
- Chloroquine
- Gold compound
- Nitrofurantoin
- Rifampicin
- Chloxacillin

Drugs Increasing Prothrombin Time

- ACTH
- Amidopyrine
- Heparin
- Morphine
- Salicylates
- Alcohol
- Chloramphenicol
- Indomethacin
- Quinidine
- Streptomycin

Drugs causing Decreased Values of Platelets

- Busalphan
- Arsenicals
- Heparin
- Quinine
- Tolbutamide
- Cyclophosphamide
- Chloroquine
- Gentamicin
- Rifampicin

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Principles and Practice of BURN CARE

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